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ON TEMPORARY PARALYSIS AFTER EPILEPTIFORM AND EPILEPTIC SEIZURES; A CONTRIBUTION TO THE STUDY OF DISSOLUTION OF THE NERVOUS SYSTEM.[1]

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In the Mirror of the 'Lancet,' December 13, 1873, and October 26, 1878, are reported, with remarks, cases of temporary local paralysis after epileptiform convulsions; after those convulsions, that is to say, in which spasm begins in the terminal part of a limb or in the side of the face: these convulsions were first described by Bravais in 1824. As I there stated, the paralysis is supposed to depend on exhaustion of nerve-fibres consequent on the excessive discharge in the paroxysm. I do not now, however, speak so confidently as to the geographical position of the fibres exhausted; physiologically speaking, they are, I suppose, those connected with the discharging cells, and are presumably some fibres of the internal capsule. Since the remarks in the first report

[1] The term Dissolution has long been used by Herbert Spencer as the opposite of Evolution. I use it in that sense in this, as I have done in former papers. Great objections are made to this application of the term, but I submit that it is inexpedient to coin a new word for the opposite of Evolution of the Nervous System, when Dissolution has been used for at least fifteen years for the opposite of Evolution in general. The term is none of my choosing; it was used as I am now using it, long before I thought of the process it names.

appeared, I have found that the hypothesis advanced is in all essential respects the hypothesis of Todd and Alexander Robertson. It is briefly that the temporary post-epileptiform paralysis depends on temporary central exhaustion consequent on the excessive discharge during the paroxysm. During the paroxysm there is a severe nervous discharge (liberation of energy) beginning from highly unstable cells ("discharging lesions"[1]) of some portion of the cerebral cortex; the currents developed of course "flow" in the order of lines from those of least to those of most resistance; in other words, the movements of the external parts represented are developed in the order of their speciality of representation by the nervous arrangements (cells and fibres) of the portion of the cortex in question. Spasm in convulsion is nothing more than a contention, or sequence of contentions, of many of the movements represented by unstable cells of some part of the cerebral cortex; numerous movements being developed in a short time.

The convulsion is the external positive event answering to the internal positive event of excessive nervous discharge (great liberation of energy). The local paralysis remaining when the convulsion is over, when the discharge has ceased, is of the parts first and most convulsed, and is supposed to be the external negative condition answering to the internal negative condition of exhaustion of some fibres overworked from the excessive discharge of cells connected with those fibres. The nerve-fibres being only exhausted, not destroyed, as in some other kinds of paralysis, post-epileptiform paralysis is temporary, unless, of course, the fit recurs soon.

The condition following an excessive cerebral discharge is supposed to be analogous to exhaustion, by strong faradization, of a nerve going to the cut-off leg of a frog. A

[1] The very simple expression "discharging lesion" has, I find, led to difficulties. It has been taken to mean that some morbid product discharges, just as the expression "the kettle boils" might be taken to mean that the copper vessel and not the water boils. A "discharging lesion" consists in some alteration (excess of function) of some part of the patient; certain of his nerve-cells, representing some movements of some parts of his body, have by some pathological process become highly unstable; on their discharge, the movements they represent are suddenly developed in great numbers in a very short time. The contention of these movements is spasm.

closer analogy is the temporary paralysis of a frog after a tetaniform attack induced by a large dose of strychnia; the functional power of the motor nerves is temporarily destroyed. Since after an enormous dose of strychnia there is paralysis without prior convulsion, it is believed that strychnia may act directly on the motor nerves. However, Vulpian concludes:[1] "Les nerfs d'un membre privé de circulation chez une grenouille strychnisée perdent donc *une partie* de leur motricité sous l'influence des excitations prolongées, répétées et violentes, qu'ils subissent dans ces conditions." A still closer analogy is that, on strong faradization of a frog from mouth to anus there is a tetanic condition, and after it a *temporary* "résolution flasque et générale de toutes les parties du corps."[2]

The above is not the accepted explanation. Some physicians believe the temporary post-epileptiform paralysis to result from the cerebral congestion of asphyxia induced by arrested respiration in the paroxysm. In reply, it may be said that such congestion being universal in the brain cannot account for *local* paralysis. Again, the paralysis has been attributed to small extravasations of blood caused by congestion from arrested respiration. But the paralysis is of the parts first and most convulsed in the prior paroxysm, and it would be very remarkable if the extravasations in each succeeding fit always occurred in the same centre—in that representing the parts first and most convulsed. I do not adduce against this opinion the fact that the paralysis is temporary, for I admit that small clots may produce only temporary paralysis. There are other strong arguments against the accepted explanation. We find local paralysis after some epileptiform seizures in which there has been no interference with respiration whatever; paralysis so occurring cannot be caused by cerebral congestion, nor by extravasations in the nervous centres. On the other hand, local paralysis does not occur after severe seizures of epilepsy proper, in which there often is extreme asphyxia from spasm of respiratory muscles. To repeat; absolute, although temporary, local paralysis may be found where there has been no asphyxia in the prior paroxysm, and is not found after paroxysms of epilepsy proper when

[1] Arch. de Phys. 1870, vol. iii. p. 128. [2] Vulpian.

the asphyxia has been suddenly induced and extreme in degree.

It is asserted by some authorities that the local paralysis after epileptiform seizures is *not* always of the parts first and most convulsed. In my experience it always has been so. This being no answer, I can only ask that further observations may be made on the matter.

It is understood, of course, that we are speaking of paralysis following seizures which we may call chronic; we are not speaking of cases of meningeal or cerebral hæmorrhage; in some cases of cerebral hæmorrhage the clot which destroys some part of the brain, and which in this way produces hemiplegia, also causes convulsion. We speak of cases such as those in which a small tumour or other "foreign body" in the mid-cortical region of the brain leads to instability of cells near it[1]—leads to what I have called a "discharging lesion"; the cells rendered unstable occasionally "explode" or liberate much energy, or, in other words, discharge excessively, and all of them much more nearly simultaneously than the comparatively stable cells do in health. After their excessive discharge has ceased, as signified by cessation of spasm, there often *is* paralysis, and that paralysis is temporary.

It is supposed that the following is the sequence from a growth in the cortex to the temporary post-epileptiform

[1] I have suggested that the size and shape of cells, as well as their nearness to the tumour, or other source of irritation, will have to do with their becoming unstable; other things equal, the same quantity of matter in many small cells will present a vastly greater surface to the contact of nutrient material than the same quantity in one large cell. I have also suggested that small muscles, or, more properly, movements which require little energy for the displacements they have to effect (those of the face and of the hands in touch, for example), are represented by small cells. Such movements are rapidly changing during many of the operations they serve in—writing, for example—and require repetitions of short liberations of energy, and necessitate quick recuperation of the cells concerned. Movements of the upper arm are, in comparison, little changing, and require persistent steady liberation of energy. That small muscles are represented by small cells is not altogether a mere hypothesis. The masterly researches of Bevan Lewis seem to me to show that those parts of Hitzig and Ferrier's region which especially represent small muscles have most small cells. It is to be noted that parts like the hand will be represented in a great number of different movements, and therefore by numerous ganglion cells. It may be mentioned, too, that epileptic and epileptiform seizures usually begin in small muscles, and it often happens that for a time a patient's seizures are limited to small muscles.

paralysis. (1) A growth, often a syphilitic one, in the cortex cerebri; so far apparently there are no convulsive or paralytic symptoms. But the growth, in its character as a foreign body, (2) induces (presumably slowly) changes of instability in nerve cells near it. Still there may be no outward signs of it, or rather of these changes it induces. Next (3) some day the unstable cells discharge and produce a convulsive paroxysm. (4) After the paroxysm there is paralysis of the parts which were first and most convulsed. (5) The patient is apparently well again in a few hours or days, and keeps so until the next fit comes. (Of course the "foreign body" may produce the general symptoms of tumour, optic neuritis, headache, &c., which do not here specially concern us.)

It is recognised nowadays, thanks especially to Hitzig, Ferrier, and Charcot, that tumours in the mid-region of the brain, when very large and when much softening is induced, may be the cause of some permanent paralysis; here the paralysis is owing to permanent destruction. In this paper temporary paralysis after convulsion is alone considered, and so far only local temporary paralysis after epileptiform seizures.

It is not denied, of course, that convulsion may occur in a part which is already paralysed, partially or completely. For example, a patient may become hemiplegic without convulsion, and then on partial, or, indeed, on apparently complete, recovery, he may become subject to convulsions beginning in the hand, side of face, or foot. This I believe happens most often when the hemiplegia has resulted by embolism or thrombosis.

Cases of transient paralysis not preceded by convulsion, however often repeated, and however much like one another the recurring palsies may be, do not come here under our notice. Transient paralysis may be owing to a small clot, or to softening from blocking of a small vessel. Repetitions of similar local paralyses, each transitory, are hard to explain; but, as above said, we are not here concerned with them. Again, a patient subject to localised convulsion may have a batch of them, and in the intervals of the convulsions the parts occasionally severely convulsed may remain absolutely

paralysed. It is very singular to witness spasm beginning in and spreading over a limb which the patient can at no time move in the slightest degree.[1] In this paper, I repeat, temporary paralysis after convulsion is alone considered.

Let us note the sequence of phenomena in a very simple case, recorded in the 'Lancet,' October 26th, 1878, intercalating hypothetical explanations in brackets, and thus keeping them separate from the facts. A patient subject to epileptiform attacks [having permanently a local "discharging lesion"] has no obvious paralysis [since if the nervous arrangements, their cells being highly unstable, are useless for normal function, there is large Compensation by neighbouring parts]. One afternoon she has a fit [an excessive discharge, beginning from the part of the cortex the cells of which are unstable], the spasm affecting chiefly the right arm [brutal, that is excessive development and more nearly simultaneous development of the movements which the unstable cells discharging represent than would occur in healthy operations]; after the fit the arm is *absolutely* paralysed [exhaustion of central nerve-fibres has been effected by the discharge]; all paralysis is gone in five hours [there being only exhaustion, recuperation is soon complete].

Of course there are cases in which the post-epileptiform paralysis is trifling, and more transitory than in the case above instanced; the patient may say his hand is a "little numb," and we may find that he grasps firmly, but cannot pick up a pin or button his shirt. It is convenient to take for illustration a simple case; the reasoning is essentially the same. In the case instanced we have not merely to account for (1) local paralysis after a localised convulsion, but for two other things also; (2) for the absoluteness of the paralysis; (3) for the paralysis being very transient.

We have so far, except in passing illustration, spoken only

[1] Perhaps it may seem that such cases are counter to the hypothesis. But that the comparatively slight discharges during "volition" should fail to move a part, and that an excessive discharge should move it, is not extraordinary. Yet if the fibres be exhausted, how is it that by them the excessive discharge can effect convulsion of the part they pass towards? We must suppose that they are either not absolutely exhausted, or that they soon recuperate enough to be affected by the excessive discharge.

of epileptiform seizures. Logically, if Todd and Robertson's hypothesis is to be sustained, there ought to be paralysis after seizures of epilepsy proper. It would usually be said that there is none. So that whilst after the comparatively slight seizures called epileptiform there is often decided paralysis, there is, according to current opinions, none after the much severer paroxysms of epilepsy proper. At first glance this seems decisive against Todd and Robertson's hypothesis, as well as against the hypothesis of congestion and extravasation already combated. But local paralysis (of the face, or of a limb, or of one side of the body) is not the sole paralysis met with: there is such a thing as paralysis spread all over the body. The patient, after a severe paroxysm of epilepsy proper, has not, it is true, any paralysis of but one limb; he is not hemiplegic. He is, however, I submit, biplegic, if such a word may be coined. I mean that there is some, however slight, paralysis of both sides of the body; the paralysis is nearly evenly, I do not say absolutely equally, spread on both sides of his body. How does it happen that such paralysis is ignored? One reason I submit is, that being slight, evenly spread, and transient—there being no difference, or no obvious difference betwixt the two sides of the body—it is not easily recognisable as paralysis. Another reason is, that here, as in other cases, abnormal physical conditions are erroneously "explained" as being owing to abnormal psychical states. After a very severe fit of epilepsy proper, the patient does not move his limbs; all admit that. He seems a mere breathing, circulatory, &c., mechanism, lying otherwise inert on the floor. As he is then comatose, it would generally be said that he does not move *because he is unconscious*. A moment's consideration, however, will show that this is an explanation which verbally explains everything, and yet in reality explains nothing. It may pair-off with such a pseudo-explanation as that a patient does not move a limb because there is loss of volition, or with that which accounts for an aphasic's inability to speak, or for his speaking badly, by saying that it is "*because* he has lost the memory of words." It belongs to a whole family of psychologico-materialistic confusions; it is akin to that which has it that ideas or sensations (psychical

states) produce movements (physical states); to that also which declares that the mind affects the body. Nothing is said against the popular or clinical use of these expressions. No one objects in a clinical conference to the expressions that ideas or emotions affect, or, more inclusively, that the mind affects the body, when it is understood that what is meant is that centres, *during activity of which mental states arise*, can affect other centres, and thus the body. But much is to be said against the use, or rather abuse, of such expressions in what purport to be scientific explanations. It is, to say the very least, inexpedient in a scientific exposition to attempt to explain physical conditions by invoking crude popular psychological doctrines.

To return to the case. I submit that the post-epileptic comatose patient does not move, for the very simple reason that there is some negative *physical condition* of his nervous centres. It is an intelligible, if it be an erroneous, explanation to say that his highest nervous arrangements, and no doubt many lower ones too, are exhausted by the excessive discharge in the prior paroxysm. Surely the patient's not moving, if it be not owing to exhaustion of nervous arrangements, is at any rate owing to *some* negative *physical* condition; this negative physical condition is not loss of consciousness, but is only correlative with that negative *psychical* condition, if one may be allowed to speak of two negative conditions being correlative. I submit that it is not an intelligible explanation to say that the patient does not move because he is unconscious. Why should he not move if unconsciousness were all, his nervous system being sound? The fact is, he cannot be unconscious without having some negative physical condition of his nervous system answering to that negative psychical condition, and it is the central negative physical condition alone which we have to take count of in our explanations of his other physical condition of immobility.

The paralysis after a paroxysm of epilepsy proper, although widespread, is not necessarily, I suppose never is, total anywhere; it varies in different cases from slight to severe. In some cases of post-epileptic coma there is considerable movement on disturbing the patient. This is not in the least

counter to the hypothesis, for besides the possibility that but few of the patient's highest nervous arrangements are exhausted, many others, and also some of lower centres, are capable of functioning. There is no *à priori* reason why there should not be slight quasi-trifling paralysis spread all over the body—in other words, loss of some movements of every part of the body, with retention of many other movements of every part of the body—than that there should be slight quasi-trifling paralysis throughout an arm as there often is—in which case there is loss of some movements of the limb as a whole, with retention of many other movements of it. To speak of a part as being both paralysed and also as being movable by the patient, is not even unusual in ordinary clinical language. For to say that an arm is partially paralysed means that there is both some paralysis and also some movement remaining. We should not speak of paralysis of *muscles* from central lesions. Nervous centres do not represent muscles, but, or except as, movements. Hemiplegia appears as loss of power in certain muscles, but really is a loss of a number of movements of the parts paralysed. Similarly a convulsion appears as spasm of muscles, but is really a development of numerous movements of the parts, or rather an attempt at such development; spasm being a contention of many movements. From a small destructive lesion of a centre a few movements of muscles may be lost, and many other movements of the same muscles be retained; the hemiplegic man, who cannot easily button his waistcoat, and yet can lift a chair, has only lost some power in the muscles of the arm, in the sense that he has lost some of the most special or "delicate" movements of this limb. Similarly there may be loss of some of the most special or "delicate" movements all over the body, with retention of very many more general or "coarser" movements all over the body.

I do not shirk the logical consequences of the doctrine that the highest centres are, like all lower centres, sensori-motor—that from loss of function of more or fewer of them there is paralysis. Let me go into detail. I believe that in every case of insanity, however slight it may be, there is, so long as it lasts, defect[1] of consciousness. I believe, too, that there is neces-

[1] I dare say it may be denied that in some slight cases of insanity there is any

sarily correlative with this negative psychical condition, a negative physical condition of the highest nervous arrangements. Putting this otherwise, consciousness in health attends, but is not activity of these highest nervous arrangements; and when the function of them, or some of them, is lost by any pathological process, there is correspondingly loss or defect of consciousness. The highest centres are not consciousness, they are only the anatomical substrata of consciousness. Since the anatomical substrata of consciousness are, like all other centres, made up of sensori-motor arrangements, there should be widespread, however slight, paralysis in every case of insanity. I believe there is. Of course, in many cases, it would be difficult or impossible to demonstrate any. Some of those who might deny there to be any in a case of insanity might nevertheless admit it by saying that there was loss of expression, shambling gait, and slowness of movement. To take an extreme case, one of temporary acute mania after an epileptic paroxysm. The furious post-epileptic maniac is unconscious as well as maniacal. His symptomatic condition is double; there is a negative and there is also a positive element. I believe there is during his mania widespread paralysis consequent on temporary exhaustion of many of his highest nervous arrangements, which exhaustion is the negative physical condition answering to his negative psychical condition. I do not mean that his maniacal actions are the outcome of the exhaustion; that is an impossible explanation; there is supposed to be paralysis answering to that exhaustion. How then do we account for the co-existing over-movements of mania? The case is an example of Dissolution, using this term as the opposite of Evolution.

Evolution does not simply imply a progressing increase of complexity and speciality only, but also that the higher (more complex and special) nervous arrangements control or inhibit

defect of consciousness, partly because it is erroneously inferred that what is clinically called insensibility is meant. Yet probably some of those who would deny defect of consciousness in a slight case of insanity would tacitly admit it by saying that the patient's judgment, power of attention, emotional control, or will was defective. A person has not got a consciousness in addition to will, memory, and emotion; these are only names applied to artificially distinguished aspects of consciousness; defective judgment, &c., is defect of consciousness.

the lower. There are two things: "the adding on" of the higher is at the same time "a keeping down" of the lower. Now as to the reverse process of Dissolution. It also is a double process. "The taking off" of the higher is a "letting go" of the lower. On removal of the influence of some of the highest nervous arrangements (on loss of their function in the case of post-epileptic mania by exhaustion), the next lower nervous arrangements, no longer controlled (Anstie, Dickson), spring into activity, and it is from *their* activity that the maniacal movements result. On the physical side there is loss of function of some of the highest nervous arrangements, and increased activity of the next lower. Correspondingly on the psychical side there is loss of consciousness and mania. In all other cases of insanity there is a negative and a positive condition, in some, as in post-epileptic mania, the positive condition is hyperpositive.[1] There is no inconsistency in supposing that there is paralysis in the sense of there being loss of some of the most special movements all over the body, and that there is also retention of very many others, and over-increased activity of them, any more than there is in supposing there to be loss of some movements of the arm in cases of chorea, where other movements of that limb are over-developed. I submit that what has

[1] I consider that there are many degrees of Dissolution consequent on exhaustion of different amounts effected by epileptic discharges of different degrees of severity. To make, perhaps with some arbitrariness, three degrees, there is (1) post-epileptic "ideation;" (2) post-epileptic action (mania for an example); and (3) post-epileptic coma. This is using ordinary nomenclature, which is misleading. There is a negative and a positive element in each degree. Along with post-epileptic ideation there is—(*a*) defect of consciousness; with the post-epileptic actions, there is (*b*) loss of consciousness, and in the third degree there is (*c*) coma. We compare these three negative conditions, saying they signify respectively Shallow, Deep, and Deepest Dissolution, the central condition being "shallow," "deep," and "deepest" exhaustion of nervous arrangements of the highest and perhaps also of lower centres. The positive elements comparable are (*a*) the "ideation," (*b*) the actions, and (*c*), not the coma, but the vital operations, circulatory, respiratory, &c., going on in the comatose state. These are owing to activity of nervous arrangements which are healthy except for being uncontrolled by their now exhausted higher nervous arrangements. Unless the duplex conditions in each be recognised, the three degrees cannot be shown to be analogous, nor can the cases be shown to come under the principle of Dissolution of the Nervous System. It is understood, too, that what we really compare and contrast are the physical conditions in the several degrees.

been called defect of voluntary co-ordination of the arm in chorea, is simply loss of so many movements of the limb, that it is so much paralysis, and what has been called morbid involuntary co-ordination, is simply over-development of some others. Besides we daily see co-existing the diametrically opposite conditions of paralysis and spasm, as in the rigidity of hemiplegia. So we do more rarely in the tremor of disseminated sclerosis, in which there is loss of some movements and retention of some others.

As aforesaid, the immobility of the post-epileptic comatose patient, which is here called paralysis, is commonly "explained" as being owing to a psychical loss. It would be denied that there is any paralysis. Yet after all, what is denied in technical terms is admitted in simple ones. On return of consciousness after the paroxysm, or of some consciousness, whilst it would be said that the patient is not paralysed, paralysis is really admitted in the statements that he is "prostrated" or "weak" after his fits. Even after slight epileptic attacks (le petit mal) there is veritable paralysis; the patient is weak ("unfit for anything," "thoroughly done up," are the kind of expressions patients use). Let me say explicitly that if paralysis is to mean only local paralysis, the patient after a slight or severe epileptic paroxysm is *not* paralysed. I admit that most willingly, indeed I assert it. But it is not fair to make such a limit to the meaning of the term. I submit that the patient is paralysed. His "weakness" is veritable paralysis, even if it be owing especially to exhaustion of nervous arrangements for vital organs (cardiac, respiratory, &c.).

I venture then to submit (1) that the psychological "explanation" is really no explanation of the comatose patient's immobility; the explanation of a materialistic condition should be in materialistic terms. Our concern, as physicians, with negative or positive psychical conditions is simply to get to know what is going wrong in the *nervous system*. The absolute distinction betwixt mind and body is insisted on, not as some seem to suppose, because a psychological view is taken of any nervous diseases, but for the diametrically opposite reason. *The distinction is made in order that we may be thoroughly materialistic in our dealings with disease*, and that

we may methodically consider the nervous system from top to bottom as a mere sensori-motor mechanism.[1] Next I submit (2) that there is paralysis after epileptic seizures and that (3) this paralysis is universally spread, and (4) is owing to exhaustion of some nervous arrangements of the highest centres and perhaps of lower ones too.

That loss of function of nervous arrangements of the highest centres (which centres, although neither they nor their activities are consciousness, are yet the anatomical substrata of consciousness), should entail paralysis in the sense of universal weakness, is in accord with the doctrine of Evolution of the nervous system. And that it should not entail local paralysis is equally in accord with that doctrine. I have long held that the very highest centres are, like all other centres, sensori-motor. In other words, parts of the brain other than Hitzig and Ferrier's centres (lower cerebral centres) are, I think, made up of sensori-motor arrangements. I do not say this in consequence of their experiments; I urged it before their experiments were begun, and urge it still. For various reasons I think that the nervous arrangements of the highest centres are the most special and complex of all sensori-motor arrangements. Each unit of the highest centres represents, I believe, the organism as a whole (complexity) and each unit represents

[1] Scientific materialism is quite a different thing from crude popular materialism. Scientific materialism distinguishes betwixt mind and nervous system in order to study each thoroughly. Popular materialism does not separate the two, and mixes up in its "explanations" psychical factors and physical factors. Scientific materialism is only materialistic as to what is material, the nervous system. Popular crude materialism, making no distinctions, confuses two utterly different things, psychical states and physical states. It is said that in an early stage of paralysis agitans the patient can keep his hand still by an act of volition. No one objects to this phrase used clinically if the physical process corresponding to what is psychically volition be meant; if it be meant, for example, that a cerebral discharge can inhibit the movements, or inhibit spinal or other centres for them. But if it be really meant that The Will can arrest a physical process, the statement is most unwarrantable. It amounts to saying that the will influences matter. Let any one try to conceive how the will can stop a movement; it is on the other hand conceivable, if not believable, that a nervous discharge *during* "volition" may stop a movement, for both are material things. Clifford writes ". . . if anybody says that the will influences matter, the statement is not untrue, but it is nonsense. The will is not a material thing, it is not a mode of material motion. Such an assertion belongs to the crude materialism of the savage."

the whole organism differently (speciality). There is, to speak figuratively, "an experiment made by disease," which seems to me to demonstrate that the highest nervous arrangements represent the whole organism. In a severe paroxysm of epilepsy proper, one setting in with loss of consciousness (when of necessity, therefore, the corresponding physical process of discharge begins in some part of the highest centres) the whole organism is involved; there is universal convulsion. The convulsion in epilepsy proper is, I take it, a brutal development of movements represented in the highest centres primarily discharged, and doubtless too in lower centres secondarily discharged. The discharge "by disease" beginning in some part of the highest centres of either side of the brain shows that they represent movements of all parts of both sides of the organism; they show this as certainly as artificially induced discharge (by galvanism or faradism) of any centre in Hitzig and Ferrier's region shows it to especially represent movements of some external part. This conclusion from an "experiment by disease" is strictly in accord with the doctrine of Evolution. As consciousness, or most vivid consciousness, represents the whole subject (subject-object) so the anatomical substrata of consciousness, or in other words the highest nervous centres, represent the whole organism. There is still another way of looking at this aspect of the question.

In many cases of epileptiform seizures beginning by discharge of some part of the mid-region of one side of the brain, and developing first local one-sided spasm, the convulsion *becomes* universal. The difference betwixt such a convulsion from discharge of part of lower cerebral centres and one of epilepsy proper from discharge of part of the highest centre, is only a difference in Compound Degree; it is such a difference as great difference in Evolution of the two centres would lead us to expect. The doctrine of evolution implies that the highest centres of one side are evolved out of the lower cerebral centres of the same side; that they re-represent all the parts which the lower centres have represented but in more numerous, more complex, and more special combinations.[1]

[1] That both sides of the body are represented in each half of the brain is quite certain as "descending" wasting in certain cases of one-sided cerebral disease

The two convulsions, (1) epileptic from discharge of part of the highest centres and (2) epileptiform, from discharge of part of lower cerebral centres, are in accord with these statements. The severe convulsion of epilepsy proper is more quickly universal, the parts on the two sides of the body are convulsed more nearly simultaneously, and the different movements of every part are developed more nearly contemporaneously, than in the severe epileptiform seizure. That it is a difference of degree is shown, in part at least, by the fact that in most cases of epilepsy proper the convulsion affects one side a little earlier and a little more than the other.

Frequently, however, the epileptiform convulsion is limited; the spasm is local. Just as after localised convulsion (epileptiform seizure) there is local paralysis, so after universal or widespread convulsion, there is, I submit, universal or widespread paralysis. The explanation given of post-epileptic immobility is, it seems to me, at any rate as good as the psychological explanation; if, indeed, the latter deserves the name of explanation.

To return now to cases of patients locally paralysed after localised epileptiform seizures. It may be alleged against the hypothesis put forward, that there is no proportion betwixt the severity of the seizure (the severity of the discharge), and the subsequent paralysis (amount of exhaustion). But once more it is pointed out, that decided local paralysis is usually what is solely called paralysis. Widespread slight temporary paralysis is not easily recognised as paralysis. If a man has total weakness of one arm, or even slight weakness of it after an epileptiform convulsion affecting that limb, that weakness is called paralysis. But if there be universal transitory slight weakness after a seizure, it is not called, but really is, paralysis.

On the hypothesis, there ought logically to be an exact relation of proportionality betwixt the discharge and the subsequent central exhaustion; betwixt the convulsion and the subsequent paralysis. I submit that there is. The relation is,

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proves. And since the fibres wasted in the spinal cord are some of those of the opposite lateral column and some of those of the anterior column of the same side as the cerebral lesion, there is warrant for the inference that the two sides of the body are differently represented in each half of the brain.

however, not easy to see at first glance. I ask the reader to remember that I expressly admit, that after the severest epileptiform seizures, there is less paralysis than after the slight ones, *if by paralysis local paralysis be meant*, and that I expressly deny that there is less if paralysis be used, as I submit it ought to be, for widespread or universal weakness as well as for local weakness after a fit.

We must, in the remainder of this paper, discard the vague word "severe" as applied to nervous discharges in disease, and speak of quantities of energy liberable and rates of liberation. Different quantities of grey matter will be unstable in different cases, and there will be differences of degree of instability of cells in different cases. Thus, in different cases there will be different quantities of energy liberable and liberated in the paroxysms. It is quite a certain thing that these are all degrees of spasm in unilaterally beginning convulsions from slight paroxysmal twitching of a few fingers, or "starting" of the big toe, to the severest universally-becoming convulsion; hence there are different quantities of energy liberable. But we must consider not only the quantity of energy liberated by discharging cells, but the rapidity of its liberation. Of course the conditions are complex, different quantities of energy will in different cases be liberated in different times. We estimate the two things respectively, but of course very roughly indeed, by the amount of spasm, and by the suddenness of onset and rapidity of spreading of spasm.

I think as a matter of observation that, other things equal, the more deliberately spasm sets in, and the more slowly it spreads, the more local it is, and also the longer it continues; and further, that the paralysis after such spasm, is correspondingly more local, is greater in degree, and more persistent. If so, we may, on Todd and Robertson's hypothesis, say of the central process, that the more deliberately the discharge begins, and the slower it is, the less widespread are the nerve-currents developed, and the longer they continue; and that consequently the post-epileptiform exhaustion of centres is more local, more complete, and more persisting. On the other hand, the more suddenly spasm sets in, and the faster it

begins to spread, the greater, I think, is the range it attains; and further, that the paralysis after it is more widespread, less in degree and more transient. If so, we may, on Todd and Robertson's hypothesis, say, that the more suddenly the discharge begins, and the more rapid it is, the more widespread are the currents developed, and the less time do they continue; and that the post-epileptiform exhaustion of centres is more widespread, less extreme, and more transient. The paralysis, in the latter cases, is *apparently* less, because it is not so localised, because it is slighter in degree, and because it soon passes off—in a word, it is not easily recognisable as paralysis, and may yet be admitted to be so, being called weakness.

Suppose two patients, in each of whom the same cortical area is affected, and so affected in one that a certain quantity of energy is occasionally liberated slowly, say in ten minutes, and so affected in the other, that an equal quantity is occasionally liberated rapidly, say in two minutes. Let us suppose that the part of the cortex represents especially movements of the arm. I suggest that the currents developed in the first case might "flow" almost exclusively to the arm, to the parts most specially represented in the part of the cortex discharged, and that in the second case they would "flow" less exclusively to the arm, but also to parts more generally represented by it, and finally to lower and collateral centres.

The rapid and sudden spreading of spasm implies a sudden rapid liberation of energy, and thus a shorter liberation of what is liberable; the currents developed will have greater force than those from a slow liberation of an equal quantity of energy. It is not asserted that nerve impulses travel faster in cases of rapid liberation than in cases of slow liberation; but that there is in the former a greater "quantity of motion" in a short time against an equal quantity in a long time in the latter. The more rapid short liberation (since currents of greater force, although over a shorter time, are developed) will overcome greater resistances than currents of less force over a larger time from slow, lengthy liberation. As will be seen, I do not use the term force as synonymous either with momentum or energy; it is, however, difficult for me to use

the term force without misgivings, since, among other reasons, it is a term used differently by different people. Supposing every molecule of a nerve to be of the same mass, and to always travel or vibrate with the same velocity, the momentum (quantity of motion) of each impulse is equal. Suppose the quantity of energy liberated to be equal in two cases, the more rapid liberation will lead to a current in which a greater number of such momenta occur in a short time, against an equal number in a long time during the slower liberation.

The movements of external parts which any centre *most* specially represents are, physiologically speaking, those united to it by lines of *least* resistance. We must bear in mind that any centre represents external parts by lines of different degrees of resistance, and no doubt often represents parts indirectly by intermediation of other centres, lower or collateral. The more force resulting from liberation of energy upon nerve fibres, the greater are the resistances which can be overcome. When greater resistances are overcome of course the wider range has the spasm, and thus the more widespread will be the subsequent paralysis.

But why is there not, after rapid discharge, as much local paralysis in the parts which were first convulsed as after the slower discharges, as well as paralysis in those parts which are next convulsed? Let me put this again. Supposing there is such a discharging lesion that there occurs a slow discharge upon an arm leaving it completely paralysed. Let us imagine, simply in order to state the question more clearly, the cells of the discharging lesion to become so much more unstable, or many more to become unstable, that in the next paroxysm the discharge is much more rapid, and that the whole body is convulsed. Why in the latter case is not the arm as much paralysed as in the former case, although there is also some slighter paralysis of the rest of the body? As a matter of fact, I submit that the paralysis is really less in degree in the part first convulsed when the spreading of spasm is rapid, and goes beyond the part, than when it is slow and is confined to that part. There is less spasm of the part first convulsed, that is, of the parts most specially represented by the cells discharging. When by more rapid

discharges the next more resisting lines (of lower or collateral centres) *are* overcome they are *then* paths of less resistance than before. The energy when more rapidly liberated by the discharging cells may be said to escape in more ways; the currents developed are less concentrated on the external part which the discharging cells most specially represent; the currents developed overcome lines of next and next greater resistance; hence parts most specially represented in the centre are less convulsed than in slow discharge, and those less specially represented more than in slow discharges.

On the other hand, in slow discharges the currents developed will keep more to lines of least of all resistance, will "flow" to parts most specially represented; being unable to overcome lines of greater resistance, they will *continue* to flow only on the lines which can be most easily overcome; thus the spasm is local and lengthy.

If the above reasoning be valid we should expect after sudden rapid short discharges, no decided local persisting paralysis, but slight widespread temporary paralysis, often so slight, so widespread, and so temporary that it is ignored as paralysis and called prostration. If, to speak roughly, as much paralysis as makes one arm useless for four hours could be spread out thin all over the body for perhaps a quarter of an hour, it would not be recognised as paralysis at all, but as weakness, &c.

No doubt, after the more rapid discharges there is really more paralysis. The "discharging lesion" is like a fulminate; by the rapidity of the discharge it overcomes the resistance of the normally unstable (comparatively stable) cells of physiologically connected centres, and the more rapidly it discharges, the more of healthy and collateral centres is it likely to overcome; it will produce effects in more ways more nearly at once. The currents developed by a rapid local discharge having overcome healthy collateral or lower centres, the energy of these centres will be added on to that of the primal liberation; there will be a compound effect from a more rapid discharge.

PISCIDIA ERYTHRINA.

BY ISAAC OTT, M.D., EASTON, PENNSYLVANIA.

THIS drug has recently come into notice as a substitute for opium in cases where pain is present, as in neuralgia. The bark of the root is used in medicine, from which a fluid extract is made. This extract has an odour greatly resembling laudanum, a reddish colour and a peculiar warm taste. The tree grows in the arid districts of Jamaica, and may attain a height of twenty to thirty feet. The bark of the tree is smooth and of a bright colour. The natives of Jamaica call the tree the "fish-catching coral tree."[1] The method of catching fish is as follows:—the leaves, twigs and bark of the roots are macerated with the residue from the distillation of rum or lime-water. Then they are put in baskets, which are dragged up and down the ponds till the narcotic principle is extracted, when the fish are stupefied, and, upon rising to the surface, are caught and eaten with impunity. The small fish only are killed by the drug. In determining the physiological action of this medicine, I used frogs and rabbits. When a rabbit received twelve drops of the fluid extract of Piscidia subcutaneously, respiration became frequent, incoordination ensued, and the pupils dilated. When twenty-four drops were given subcutaneously, sensibility was blunted. When another dose of twenty-four drops was given subcutaneously, the strongest pinching did not move him; lay sleeping; saliva flowed; death ensued by paralysis of the respiratory apparatus—heart beating at the time of death one hundred and sixty-eight per minute. To determine the action of the drug on frogs and the circulation in rabbits, I added to

[1] 'New Remedies,' 1880.

an ounce of the fluid extract an equal quantity of warm water, and evaporated the whole quantity down to about five drachms. The temperature of evaporation did not exceed 175°. The object was to get rid of the alcohol, which would confuse the results. I shall allude to the product obtained by evaporation as an "infusion" of the drug. When a frog receives twenty drops of an infusion of Piscidia, he soon assumes a squatting posture, can be handled without any effort to escape, pupils are dilated, seems asleep, sensibility blunted. When twelve drops more of the infusion are given, he is unable to hop away; when pinched, tetanus supervenes; does not make any reflex movement upon pinching; death ensues. When the sciatic nerve is exposed, it is irritable to a very weak current. To study the effect on the sensory nerves I used the ligature. For reflex action, the method of Türck was employed. Upon the circulation, the changes were studied by means of Ludwig's kymographion. The results obtained by these methods are found in the table on page 454, where they are compared with those of morphia and chloral.

Compared with atropia, piscidia, unlike it, does not paralyse the motor nerves, it does not paralyse the chorda tympani; it does not arrest the sudoral secretion; it does not paralyse the pneumogastric; it does not elevate greatly the arterial tension, but, like it, dilates the pupil. In its action on a man in health, it reduces the pulse, causes salivation and sweating, disturbance of vision, itching of skin, sleep. It seems to be more powerful to relieve pain than to narcotise. In a case of aneurism of the abdominal aorta under my care this drug acted admirably in relieving the neuralgic pains without constipating or disturbing the appetite, two things which opium did. It sometimes, when first given, causes a slight nausea. To obviate this, it should be given on a full stomach, and Dr. F. L. Putt[1] recommends its combination with a small amount of glycerine, camphor-water, and a few drops of essence of wintergreen in syrup. The nausea that ensues after opium has been taken is seldom seen after the action of Piscidia. Neither does Piscidia leave a

[1] 'Therapeutic Gazette,' Sept. 1880.

general nervous disturbance, like opium. As a sedative, for the cough of different pulmonary complaints it has been found quite useful. That this drug acts somewhat peculiarly, due

Piscidia Erythrina.	Morphia.	Chloral.
It is narcotic.	It is narcotic.	It is narcotic.
It does not affect the irritability of the motor nerves.	It increases and then decreases the irritability of the motor nerves.	It does not affect the motor nerves.
It does not act on the peripheral ends of the sensory nerves.	It excites sensory nerves.	It does not affect sensory nerves.
It reduces reflex action on the centres of Setschenow.	It increases and then reduces reflex action.	It increases and then decreases it.
It produces tetanus by a stimulant action on the spinal cord.	It causes tetanus by a spinal stimulant action.	It convulses.
It dilates the pupil.	It contracts the pupil.	It contracts the pupil.
It is a salivator.	It dries up salivary secretion.	
It excites sudoral secretion.	It causes slight sweating.	
It reduces the frequency of the pulse.	Small doses slow the pulse and then accelerate it; large doses slow the pulse by an action on the heart.	It reduces the frequency of the pulse by an action on the heart itself.
It increases arterial tension by stimulation of the monarchical vaso-motor centre; this rise of pressure is soon succeeded by a fall, due to an action on the heart and the vaso-motor centre.	It increases and then decreases the arterial tension, due to stimulation and paralysis of the main vaso-motor centre.	It reduces arterial tension by paralysis of the main vaso-motor centre.

probably to the idiosyncrasy of the patient, is shown in a case reported to me.[1] A man with paraplegia, supposed to be syphilitic, was given at 8.30 P.M. a drachm of the liquid-extract

[1] Letter of Dr. C. E. Bean, Jefferson Medical College Hospital.

of Piscidia, which produced profuse sweating, with increased action of the heart, the pulse beating stronger and fuller, followed by a rapid and weak pulse. At 10.30 P.M. the dose was repeated, the same effect ensuing. At midnight a third drachm was given, and in about fifteen minutes the skin grew quite cold, respiration laboured, and pulse very feeble. He lay in a stupor two hours, after which he slept an hour. Repetition of doses of a drachm or two drachms on following nights caused no narcotic effect, but perspiration, nausea, with loss of appetite in the morning. From my knowledge of this drug in its physiological aspect,[1] and in the practice of my profession, I regard it as well worthy of a trial by the profession. The consensus of several good observers is needed to establish the proper therapeutic rank of this agent.

[1] 'Detroit Lancet,' June 1880.

CEREBRAL AMBLYOPIA AND HEMIOPIA.[1]

BY DAVID FERRIER, M.D., F.R.S.

Some important differences of opinion still exist respecting the relations of the eyes to the cerebral hemispheres, and the facts of clinical medicine and pathology appear in some respects neither to agree altogether with any of the views founded on physiological experiment, nor to be quite in accordance with each other. My object in this communication is to remove some of these differences, and to endeavour to explain some of the apparent discrepancies among clinical facts. As regards the relations of the eyes to the cerebral hemispheres, and the localisation of a visual centre, the question may be briefly stated to stand at present thus :—

In monkeys and in other animals, I, followed by Munk, Luciani and Tamburini and others, have found experimentally that destructive lesions of certain parts of the cortex cerebri cause affections of vision, and of vision only, other forms of sensibility and motor powers remaining intact. In monkeys I localised[2] the visual centre in the angular gyrus; and I have stated that unilateral destruction of the angular gyrus causes total loss of vision in the opposite eye, of a temporary character, however; while bilateral destruction entails total loss of vision in both eyes—an effect which I inferred would be of a permanent character. I also stated that the greater portion of both occipital lobes might be removed without any discoverable impairment of vision.

Munk[3] localises the visual centres in the occipital lobes.

[1] The substance of this Paper was read at the Meeting of the Brit. Med. Ass. at Cambridge, Aug. 1880.

[2] 'Functions of the Brain,' § 65.

[3] 'Verhandl. der Physiolog. Gesellsch. zu Berlin,' April 12, 1878.

He states that a destructive lesion of one occipital lobe causes hemiopia of both eyes—the retinæ being paralysed on the side of lesion, and therefore entailing blindness towards the side opposite the lesion. Bilateral lesions cause total blindness.

Luciani and Tamburini [1] agree with me, in opposition to Munk, in placing the visual centre in the angular gyrus; but they include also the occipital lobe, partly or wholly. They agree with Munk in stating that a unilateral lesion of the visual centre causes bilateral hemiopia, and not crossed amaurosis such as I have described. This is a very brief statement of the different views, but essentially it is all that needs be mentioned in the present relation. Goltz's facts,[2] apart from his views as to the localisation of function, also demonstrate affections of vision from cortical lesions.

Now if we turn from physiological to clinical and pathological observation, we not only have no clear evidence of impairment of vision from purely cortical lesion, but we have actually many cases on record, in which post-mortem examination has revealed destructive lesions in the angular gyrus, and in one or both occipital lobes, without any affection of vision having been observed during life.[3]

The clinical history and morbid anatomy of cortical lesions, therefore, if it does not oppose, at least does not give direct support to the localisation of a visual centre in the regions indicated. The evidence in favour of a cortical visual centre in man is mainly of a constructive character; that is to say, there are many clinical phenomena which are best explained by the hypothesis of such a centre, but they can scarcely be taken as at all clearly indicating its position.

Among the facts of this nature may be mentioned, the irritative symptoms, ocular spectra, coloured vision, &c., which occur in connection with "discharging lesions," situated towards the posterior aspect of the hemisphere; as in cases reported by Hughlings-Jackson and Gowers.[4]

[1] 'Sui Centri Psico-Sensori Corticali,' 1879.
[2] 'Pflüger's Archiv,' xx. 1.
[3] See the author's 'Localisation of Cerebral Disease,' p. 110 *et seq.*
[4] 'Lancet,' March 1879.

Secondly: the phenomena of "word-blindness" occurring in connection with lesion in the region of the angular gyrus, of the left hemisphere especially; as in a case related by Broadbent.[1]

Thirdly; the temporary abolition of vision in one eye in connection with meningo-encephalitis of the opposite hemisphere, as in a case related by Abercrombie.[2] With this may be taken also the unilateral affections of vision described by Fürstner in general paralytics.[3]

The facts relating to injury and disease of the brain in the occipital region are discussed subsequently.

Other considerations might be advanced in support of the hypothesis of a definite visual centre in the cortex; but I do not propose to enter farther into this question, which I have already discussed elsewhere.[4]

But if the relation between visual defects and lesions purely cortical is not in all respects sufficiently well demonstrated, it is otherwise as regards the effects of lesions in the internal capsule, and in the medullary fibres in the neighbourhood of the optic thalamus, corpora geniculata, and occipital region.

In cerebral hemianæsthesia of organic origin, depending, as was first indicated by Türck, on lesion of the posterior segment of the internal capsule external to the optic thalamus, and since abundantly confirmed by numerous other reported cases, vision, as well as other forms of sensibility, is affected in a very distinct and special manner.

In the classical form of this affection vision is greatly impaired, if not temporarily entirely abolished, on the side opposite the lesion; the field of vision is greatly contracted; and there is achromatopsy, or impaired colour-perception.

Generally also, as was first pointed out by Landolt, there is some contraction of the field of vision on the same side as the lesion; but the visual impairment, i.e. the diminished acuity of vision and dyschromatopsy is essentially a crossed one. I might quote many accurately investigated instances of this

[1] 'Med. Chir. Trans.' 1872.
[2] 'Dis. Brain and Sp. Cord,' p. 121.
[3] 'Archiv für Psychiatrie,' 1877 and 1879.
[4] Op. cit.

affection now on record, but it will suffice if I refer to the perimeters of two well-marked cases recently under the care of Dr. Hughlings-Jackson and myself, and notes taken by Dr. Beevor.

Case I.—T. H., aged 67, a house-painter. Never suffered from lead-colic or wrist-drop. No history of syphilis. Has had gout in the left foot three or four times.

The present illness came on suddenly seven months before admission into the National Hospital for Paralysed and Epileptic on March 30, 1880. He was engaged melting some lead, when he suddenly lost consciousness and fell down, without having had any premonitory symptoms. He regained consciousness in a few minutes, and found that he was paralysed on the right side of the body. His speech was indistinct, but he could understand what was said to him, and make himself understood in words. He says he lost both motor power and sensibility on the right side.

He first began to be able to move his fingers about a fortnight after the onset. In another fortnight he could move the elbow, and a little later he could move his arm at the shoulder.

He could not move the leg at all for two months. He first was able to flex the thigh, then the foot, then the leg, and lastly the toes.

Present Condition.—Patient is a strong, healthy-looking man. There is no cardiac disease, and the urine is free from albumen. Arcus senilis is well marked, and the arteries are somewhat rigid and tortuous.

Motility.—The right angle of the mouth acts a little less vigorously than the left, but the difference is not marked.

All the movements of the right hand and arm are possible, though feebler than on the left. The grasp of the right hand= 2 kilos., that of the left=25 kil. There is, however, some stiffness of the phalangeal joints of the right hand, which impedes firm closure of the fist. There is a good deal of fibrillar tremor of the muscles of the right arm, especially the extensors, and there is well-marked oscillation—of the disseminated sclerosis type—when he endeavours to place his forefinger on a given mark.

The movements of the right leg are all capable of being carried out, though with much less vigour than the left.

Patellar tendon-reflex is exaggerated on the right side. There is no ankle clonus.

Sensibility.—Sensibility, both general and special, is impaired on the whole of the right side.

Face.—There is complete abolition of sense of impressions of

contact on the right side of the head and face. Painful impressions are felt only on the face and head in front of a line drawn vertically through the auditory meatus. Pressure over the exit of the branches of the fifth, causes severe pain. The ear may be pinched severely without causing any sensation.

Arm.—He cannot distinguish two points on the right hand. Tactile and painful impressions on the forearm are referred to the palm of the hand. Painful impressions, as such, are recognised and correctly localised on the fingers.

Leg.—Tactile impressions are not recognised anywhere on the leg or foot, but painful impressions are felt and fairly localised.

Muscular Sense.—Tested with balls alike in appearance, but of different weights, he can distinguish between heavier and lighter balls; but underestimates the absolute weight with the right hand, on the average making the same weight with the left hand double what he makes it with the right.

Smell is completely abolished in the right nostril. There is also almost complete anæsthesia in the mucous membrane of the right nostril.

Taste is abolished on the right half of the tongue. There is also tactile anæsthesia of this side, and of the mucous membrane of the right half of the mouth and palate.

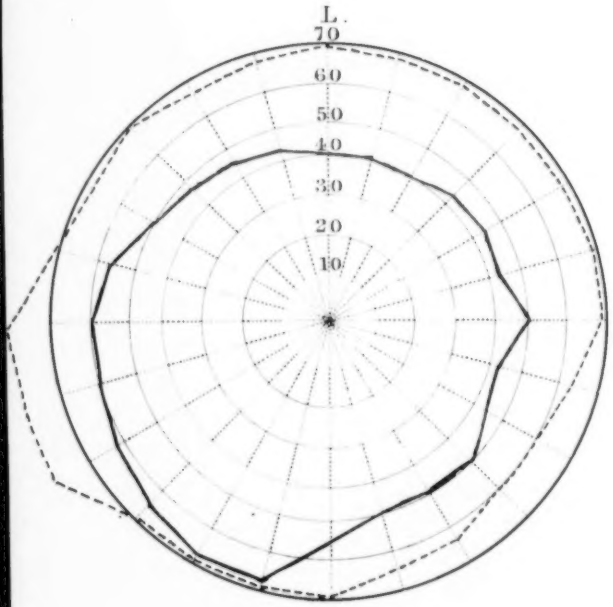
Hearing is defective on the right side. With Gellé's aural tube, he hears a watch at 15 inches on the left side, and at 8½ inches on the right.

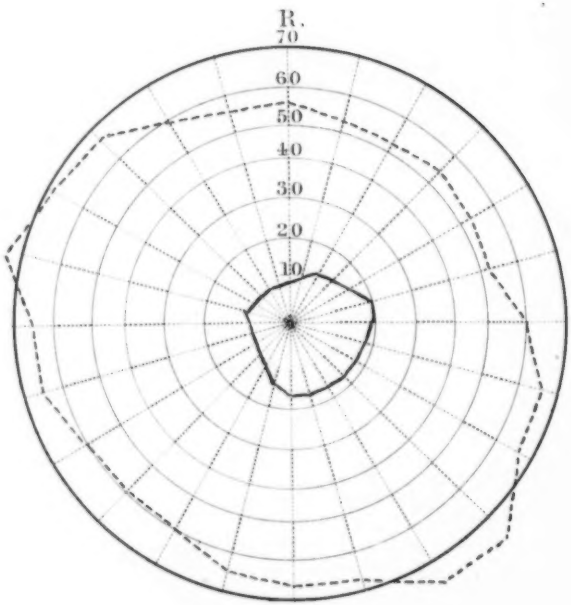
Vision.—Acuity of vision normal in left eye.

Vision is dim and indistinct in the right eye. Tested with Snellen's test types, he recognises E. No. CC. at 3 feet; C. (No. C.) at 2 feet, and LXX. at 1 foot. He requires to move his head in order to take in the whole of E., as he cannot see it all at once. Holmgren's coloured skeins he recognises and names fairly correctly.

The perimeter (see Plate I.) taken on April 24 shows that the field of vision is contracted on the right side to an almost central area—slightly towards the right—measuring from 10–15 degrees. The field of vision in the left eye measures 50 degrees from the centre towards the right, and 60 degrees to the extreme point of the left. (The want of exact symmetry depends on the prominence of the nose.)

On June 5, along with slight improvement in respect to the





C. Berjeau.

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other defects, the perimeter showed that the field of vision in the right eye had extended to 60 degrees from the centre all round, and slightly farther upwards and to the left and downwards and to the right. In the left eye the area had extended to 70 degrees all round—the limit of the normal chart.

The ophthalmoscopic appearances of both eyes were in every respect normal.

Case II.—Hannah O., aged 40, married. Has had eight children, five of whom are now alive. Four years ago, when in a state of anxiety about her daughter who was very ill, she went to bed quite well one night, but had an attack in the night in which she became insensible.

Was confined to bed for a week, and was more or less ill for six weeks, but not paralysed as to any of her limbs.

At the end of March, 1880, after a heavy day's work in the kitchen, she felt giddy in going upstairs. Shortly after she became insensible, and continued so for four days. When she came to herself, found she had lost feeling on the left side, and could not move the left arm or leg. This continued for a fortnight, at the end of which she began to move the fingers a little. The foot recovered later, but she was not able to walk without support for five or six weeks. During this time, she accidentally discovered one day, on closing her right eye, that she could not see with the left eye. Since her last attack she has continued feeble on the left side, particularly the leg, the condition of which she alone complained of when she came to the National Hospital for Paralysed and Epileptic on June 4, 1880.

Present State.—Patient is a fresh-looking woman. There is no cardiac disease, and the urine is free from albumen.

Motility.—The left side of the face acts very feebly as compared with the right.

The movements of the left arm and hand are feeble. Grasp of left hand = 5 kilos., that of the right = 25 kilos.

The left leg is very feeble, and the movements of the foot paralysed. In walking, she rubs the toe of her left boot on the ground.

Sensibility.—Muscular sense. Has a very imperfect appreciation of the position of her left arm when her eyes are shut. When she was asked to touch her nose with her left hand, she made an effort, and thought she had done it, though the arm, being firmly held was not moved. When tested with differently weighted balls, she

estimated 20 oz. in the left hand, as of exactly the same weight as 3 oz. in the right.

Cutaneous Sensibility.—She cannot feel a slight touch (such as is distinctly felt on right side) anywhere on left side. With stronger pressure, she feels and can localise fairly well, but she does not complain of pain on pinching or pricking deeply. Powerful stimuli are felt as mere contact. She cannot appreciate two points at the greatest distance possible, on the face, or hand, or foot.

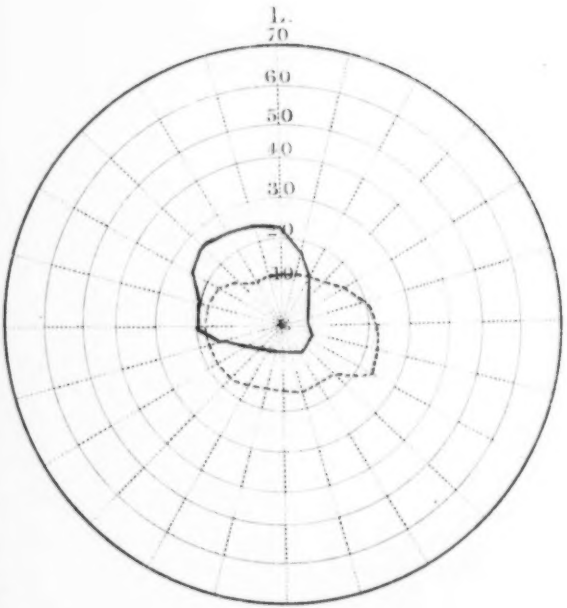
Smell.—Smell is entirely abolished in the left nostril. The mucous membrane is also anæsthetic.

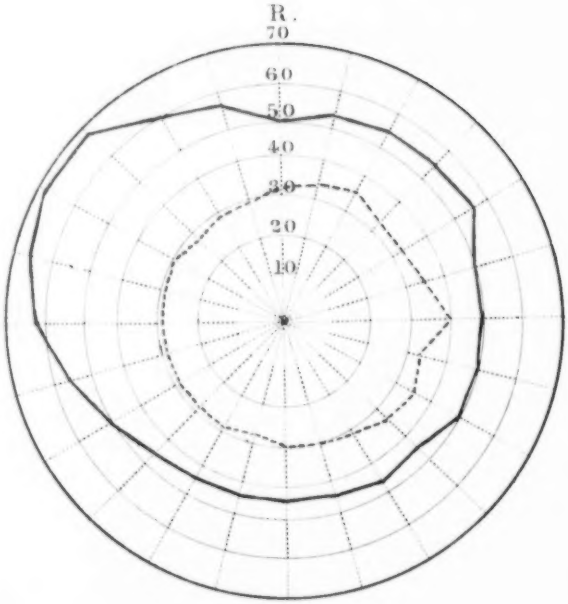
Taste.—Taste is completely abolished on the left half of the tongue. There is also abolition of tactile sensibility on the left half of the tongue, and on the left side of mouth and palate.

Hearing.—Is entirely abolished in the left ear. She cannot hear a watch or tuning-fork with this ear, either closely applied to the ear or conveyed through the skull. With the right ear she hears (through Gellé's tube), the tick of a watch at 60 cm.

Vision.—There is no ptosis. The refractive media and ocular tension normal, and the fundus and optic discs normal in both eyes. The conjunctiva of the left somewhat anæsthetic. The acuity of vision and colour-sense are normal in the right eye. Vision with the left eye is very dim. She requires to bring objects quite close up to her eye to appreciate them at all. Can only recognise Snellen CC. quite close to the eye. The colour-sense in the left eye is unusual. She can only recognise green. All other colours are described as only more or less 'glimmering.' Red in particular glimmers. This test was repeated several times afterwards, with precisely the same result. Usually, according to Charcot and Landolt, green is the next colour after violet which is most readily lost, while yellow and blue may continue to be recognised. Here, however, green only remained—a fact carefully verified by repeated and varied tests.

The extent of the visual field as indicated by the perimeter (Plate II.) taken on June 11, shows that in the left eye the visual field is contracted to an area of about 15 radius, situated somewhat above and to the left of the centre; while that of the right eye, also contracted, has a radius of 50, increasing to nearly 70 upwards and to the left. On July 30, while the





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area in the left eye continued almost exactly as before, the right had become contracted to about 30.

A re-examination of this patient on Dec. 3, showed that the symptoms had become rather intensified than the reverse, the patient generally more feeble. The visual fields were seen to be even more contracted than on July 30. The acuity of vision and colour-sense were perfectly normal in the right, while the amblyopia had become still more marked in the left. The only colour recognisable was green, which, however, she now termed blue, instead of green as before.

The disposition of the optic tracts in the chiasma, and their relations to the eyes on the one hand, and their centres on the other, have been the subject of numerous investigations, anatomical, experimental and clinical, the results of which are more or less discordant.[1] On grounds of anatomical and microscopical investigation, Biesiadecki,[2] Mandelstamm,[3] Michel,[4] Schwalbe,[5] support the theory of total decussation of the optic tracts.

Gudden,[6] by tracing the degeneration of the tracts resulting from extirpation of the eyeball, and Nicati,[7] by experimental division of the optic chiasma, support the theory of semi-decussation in all the higher animals; some of the lower, as well as fishes and birds, having complete decussation.

But clinical observation in man places the fact of semi-decussation of the optic tracts beyond all doubt. Many instances of hemiopia from lesions of one optic tract might be quoted. A case reported by Gowers[8] shows this in a very satisfactory manner, and Baumgarten[9] has demonstrated the degeneration of both optic tracts in consequence of destruction of one eye. Cases recorded by Hughlings-Jackson,[10] Remy,[11] and J. L. Prevost,[12] show also that lesions in the region of the corpora geniculata produce a similar result.

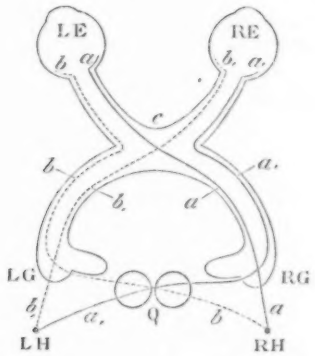
The general disposition of the fibres of the optic tracts,

[1] See Bellouard, 'L'Hémianopsie,' Paris, 1880.
[2] 'Sitzber. d. Wien. Acad.,' Bd. xlii. 1860.
[3] 'Archiv für Ophth.,' Bd. xix. 1873.
[4] *Ibid.*
[5] Von Graefe and Saemisch's Handb. 1874.
[6] 'Archiv für Ophth.,' Bd. xx. 1874.
[7] 'Archiv d. Physiologie,' 1878.
[8] 'Centralb. f. die Med. Wissensch.' 1878.
[9] *Ibid.*
[10] 'Lond. Hosp. Rep.' vol. viii. 1875.
[11] Bull. Soc. Anat. 1875.
[12] Bull. Soc. Suisse, 1879.

apart from the relative position of the decussating and non-decussating fibres, may therefore be regarded as having been satisfactorily determined.

But when we pass from these to the cortical relations, we again meet with difficulties and discrepancies.

Charcot, founding mainly on the facts of cerebral hemianæsthesia, considers, as will be seen by reference to his scheme, that the decussation in the chiasma is again reversed,



L E, and R E, left and right eyes; C, chiasma; L G, and R G, left and right geniculate bodies; Q, corpora quadrigemina; L H, and R H, left and right centres of vision; *b* and *a* nerve-fibres from left and right sides respectively of left eye; b^1 and a^1, corresponding fibres from right eye.

probably in the corpora quadrigemina; so that by this the whole of the fibres of one eye are brought into relation with the opposite cerebral hemisphere. Hence the crossed amblyopia characteristic of cerebral hemianæsthesia.

How well this scheme suffices to account for the amblyopia of cerebral hemianæsthesia will be at once apparent, and the facts which I observed in monkeys after destruction of the angular gyrus on one side led me to consider this scheme as exclusively correct. But the recent researches of my colleague Prof. Gerald Yeo and myself—communicated to the Physiological Section of the Brit. Med. Association, 1880[1]—as well as recent clinical observations, render necessary some modifi-

[1] See Abstract in 'BRAIN,' No. XI. p. 419.

cation of this, as well as other previously formulated views on the relations of the eyes to the cortical centres.

First, with respect to the negative or latent effects of lesions of the visual centres in man, viz. the angular gyri (with supramarginal lobules) and the occipital lobes.

We have found in monkeys that the only lesion which is capable of producing complete and permanent loss of vision —if even yet we are warranted in saying so much—is total destruction of the angular gyri and occipital lobes on both sides.

We have found that the effects of all other lesions are more or less transient, and that an animal can see with both eyes if only one angular gyrus remains; and still more remarkable that one occipital lobe is sufficient for this purpose. When we say that the animal sees, we do not merely mean that the eyes are sensitive to light, but that the animal can direct all its actions, and interpret, and act on, its visual sensations in an intelligent manner. We do not pretend to be able, from a study of even the most human of the lower animals, to exclude any affection of vision in its intellectual aspects; but we cannot find that the animal's modes of activity and habits are appreciably altered.

This extraordinary result will, if we may assume a complete physiological as well as anatomical homology between man and the monkey, serve to explain the absence of any permanent loss of vision in connection with lesions which do not destroy the whole area of the visual centres. I do not know of any such case in clinical records, and an event of this kind is not likely to occur under conditions in which it would be possible to arrive at really trustworthy conclusions, on account of the profound impairment of all the cerebral functions which would almost certainly be present.

The effects of unilateral destruction of the angular gyrus speedily disappear in the monkey. Hence in man, especially as the lesions of disease are more commonly only partial, or slowly progressive, the absence of symptoms in the sphere of vision occasions no great difficulty. Bilateral destruction of the occipital lobes (taking care not to trench on the parieto-occipital fissure and the posterior limb of the angular gyrus)

we have found, as I did formerly, to be without discoverable effect on vision. Hence the absence of visual defects in the cases of unilateral and bilateral occipital lesions which have been reported in man[1] is also in harmony with our experimental results.

Next as to the temporary effects of lesions of the visual centres in the monkey.

We have observed, in confirmation of my previously published results, a temporary total loss of vision in the opposite eye when the angular gyrus was completely destroyed. The total loss of vision, however, lasts at most only a few hours, after which period the modes of action of the animal undoubtedly indicate vision, to such an extent at least as to guide its movements and enable it to lay its hand on articles of food. That a condition of amblyopia may exist, however, is possible, though the determination of such a defect is in the monkey practically impossible. And the facts of hemianæsthesia show that there may be considerable amblyopia and contraction of the visual field without the patient being specially aware of it or complaining of defect of vision (see Case II.). If the lesion of the angular gyrus is not complete, the affection of vision of the opposite eye is less marked and more transient. These facts might be taken to indicate that central vision at least is possible, notwithstanding the removal of the opposite angular gyrus.

Recovery is not explicable by the activity of the other angular gyrus only, for we found that the removal of this also, after an interval of some weeks, produced only transient effect. It is different, however, when both angular gyri are destroyed simultaneously. Total blindness ensues; but on the third day there are indications of vision, though as long as three or four weeks afterwards there is still some visual defect, evidenced by the uncertainty and want of precision of aim when the animal tries to pick up articles of food lying on the floor. Extensive lesions, or even bodily removal of the greater portion of one or even both occipital lobes, cause no discoverable impairment of vision.

If, however, the angular gyrus and occipital lobe are

[1] 'Localisation of Cerebral Disease,' p. 116.

destroyed together on one side, vision is distinctly affected in both eyes. A condition of hemiopia ensues; vision being defective or abolished towards the side opposite the lesion, from paralysis of both retinæ on the corresponding side. Whether this hemiopia is symmetrical, or whether each retina is paralysed precisely on one half, or whether one eye is affected more than the other, is very difficult to determine accurately; but we should not pronounce definitively on this point. This condition of hemiopia is also only of temporary duration, though it lasts longer than the cross amaurosis from destruction of the angular gyrus. At the end of a week there is evidence of vision to both sides, and the animal soon recovers, to all appearance perfectly.

From these facts it would appear, therefore, that there is a twofold relation between the eyes and the cortical visual centres; the one mainly cross—the central portion of the retina probably bilaterally represented—by the angular gyrus; the other bilateral—the corresponding side of both retinæ being represented—by the occipital lobe, not alone, however, but in conjunction with the angular gyrus. Perfect vision is possible with the angular gyri when the occipital lobes have been removed almost up to the parieto-occipital fissure, directly after the operation. Perfect vision is also possible by the occipital lobes alone at a relatively short period after the extirpation of the angular gyri; and not only so, but even one angular gyrus, or one occipital lobe may suffice.

Many important questions as to the theory of vision and the comparative relations and development of the homologues of the angular gyri and occipital lobes in different animals are suggested by these results; but at present some of the clinical bearings only will be considered.

The cross amaurosis or amblyopia which we have found to result from lesion of the angular gyrus is entirely in harmony with the affection of vision which is usually seen in cerebral hemianæsthesia. It would therefore appear that the fibres which pass to the cortex in the posterior third of the internal capsule (posterior segment) are those which are specially in relation with the angular gyrus, and represent the opposite

eye as regards those functions which are affected in hemianæsthesia.

That a cross affection of the vision may occur from cerebral lesion seems therefore established both experimentally and on clinical grounds. Nothnagel also [1] regards this as a necessary corollary from the cases already on record. But in addition to—and by some considered incompatible with—this cross affection, the occurrence of hemiopia from cerebral lesion, especially in relation with lesions involving, if not confined to, the occipital lobe and its medullary connections, has been of late strongly urged on grounds of clinical and pathological observation [2] in combination with the physiological experiments of Munk. The cases, however, which have been so far recorded, are, taken by themselves, not sufficiently definite to establish an indisputable causal relationship between the hemiopia and the cerebral lesion as such, or to indicate with any degree of precision the locality of the centre in question. The lesions have either been multiple,[3] or of the nature of tumour,[4] or if of the nature of softening, the lesion has generally been so diffuse and extended so deeply into the medullary substance, that great caution requires to be exercised in founding conclusions on them. This is all the more necessary, inasmuch as lesion or pressure on the optic tract alone causes hemiopia, and this may be affected indirectly though the lesion is not directly situated on its course. The cases also already alluded to, reported by Hughlings-Jackson, Remy, and Prevost, show that lesions in the posterior aspect of the optic thalamus and corpora geniculata cause hemiopia. Hence the possibility of affections of these structures, directly or indirectly, must be excluded before we can consider the causal relationship between hemiopia, and the cortical or subcortical lesions is at all satisfactorily established.

Taken, however, with the facts of experiment above described, the clinical cases have a much greater value. The

[1] 'Topische Diagnostik des Gehekrankheiten,' p. 499.

[2] See Bellouard, op. cit.; and Nothnagel, op. cit.

[3] Cases by Hjort, Förster, Hosch, Chaillon (cited by Bellouard, op. cit.). Huguenin (cited by Nothnagel)

[4] Cases by Pooley, Hirschberg, Jastrowitz, Mohr (op. cit.).

following cases may be selected as the least complicated examples of hemiopia from cerebral lesions.

Wernicke's case [1] was one of right hemiopia, aphasia, and subsequent hemiplegia. There was a cortical lesion of the left hemisphere involving the angular gyrus, a considerable extent of the occipital lobe, and the upper portions of the sphenoid-temporal convolutions as far as the fissure of Sylvius. The lesion extended into the medullary fibres of the ascending parietal convolution, and deeply into the medullary substance of the occipital lobe, dividing the radiations of the optic tract. There was also a focus of softening in the left corpus striatum, but the optic thalami, corpora geniculata and quadrigemina, optic tracts and the rest of the hemisphere presented no abnormal appearance.

Baumgarten's case [2] of left hemiopia had among other slight lesions (one in the roof of the anterior horn of the left ventricle, another in the right optic thalamus), an extensive apoplectic cyst in the right occipital lobe. The rest of the brain was described as normal.

Curschmann's case [3] was one of left hemiopia, occurring without other cerebral symptoms, in the course of fatal poisoning by sulphuric acid. After death there was found "a large softening in the right occipital lobe reaching to the surface, chiefly towards the posterior and inner aspect." The optic tracts and rest of the brain were described as normal.

This appears to be the least uncomplicated case as yet on record, so far as I have been able to discover.

If we may accept these cases as being purely subcortical, and exclude all implication, direct or indirect, of the corpora geniculata and optic tracts, we may interpret them according to what has preceded, as being destructive of the radiations of the optic tract, not merely to the occipital convolutions but also to the angular gyrus. The extent of damage, even though it may appear that the occipital lobe—and this term is used as a rule too vaguely—alone is the seat of lesion, can rarely be determined accurately from the lesions of disease only.

[1] 'Verhandl. d. Physiolog. Gesellsch. zu Berlin,' April 12, 1878.
[2] 'Centralbl. f. die Med. Wissensch.,' No. 21, 1878.
[3] 'Centralbl. f. pr. Augenheilkunde,' June, 1879.

Some interesting cases are also on record of hemiopia resulting from traumatic injuries, more especially in the occipital region.

In one of these reported by Cohn,[1] in consequence of a fall on the head there was right hemiopia and some degree of aphasia for three months after the injury. Several months later, however, both the hemiopia and the aphasia had completely disappeared.

In others, again, the hemiopia has been apparently permanent. The most uncomplicated case of this kind perhaps is the one described by Keen and Thomson,[2] and also by Dr. Otis in vol. i. p. 206 of the 'Medical and Surgical History of the War of the Rebellion,' with a chromo-lithographic illustration of the seat and extent of the injury.

Patrick Hughes received a gunshot wound of the head at the battle of Antietam on September 17, 1862. The descriptions of the course of the bullet and extent of the injury given by different surgeons differ somewhat, but the description given by Keen and Thomson, with which the chromo-lithograph appears to agree, is as follows: "Wound of entrance in the middle line, $1\frac{1}{4}$ inch above external occipital protuberance—small depressed wound; wound of exit 2 by $2\frac{1}{2}$ inches, its centre being 2 inches to the left of the middle line, and 3 inches above the wound of entrance."

The man was paralysed in the right arm and leg for some time, and considerable fungus cerebri formed and was removed. He, however, recovered within a year, and was able to work. At the date of examination (Dec. 20, 1870), by Drs. Keen and Thomson, well-marked right hemiopia existed, without any pathological appearance in the fundus oculi.

This case is a valuable one, both on account of the nature of the injury and the care with which it has been investigated.

The seat of lesion and the direction of the projectile render it probable that the damage was confined to the posterior lobe of the left hemisphere, touching on, but not permanently destroying the centres for the arm and leg, situated somewhat in advance at the upper extremity of the fissure of Rolando.

[1] Cited by Bellouard, op. cit.

[2] 'Trans. of the Americ. Ophth. Soc.' July 1871, p. 122.

In many cases of injury to the head it is much less easy to exclude direct implication of the optic tract at the base, and this must always be regarded as a possible occurrence, even though the injury might appear to be purely cerebral.

Thus it is highly probable that pressure on the optic tract is the cause of the temporary hemiopia described by Gowers[1] in connection with apoplectic extravasations, existing along with conjugate deviation of the eyes. The hemiopia is towards the side opposite the lesion. We may suppose that there is, for the time being, pressure on the optic tract on the side of the effusion. This at least would seem to be the cause, unless it can be shown that other forms of sensibility, general and special, on the opposite side are also for the time abolished; facts which would indicate rather temporary annihilation of the functions of the whole hemisphere.

That a cerebral lesion in the occipito-angular region may cause hemiopia altogether apart from direct or indirect implication of the optic tract or corpora geniculata is, however, possible, is rendered clear by the experimental facts above recorded, and some of the clinical cases are best explained on this hypothesis.

A temporary functional disturbance of this region appears also most satisfactorily to account for the scintillant scotoma, so frequent in connection with migraine, the phenomena of which have been minutely described by Airy,[2] Liveing,[3] Dianoux,[4] and others.

Sometimes only one eye is affected, wholly or partially, at other times a fugacious homonymous hemiopia is observed.

In addition to the ocular symptoms, there are not unfrequently other paræsthesiæ and parakineses on the hemiopic side, with temporary disturbances of speech of the aphasic type. These phenomena, with the accompanying acute hemicrania, may all be accounted for by neurosis of the cerebral membranes, and temporary disturbance of the functions of the subjacent cortical centres.

If we consider it sufficiently well established by the above

[1] 'Brit. Med. Journal,' Nov. 24, 1877.
[2] Proc. Roy. Soc. Feb. 1870.
[3] Megrim, 'Sick Headache,' &c., 1873.
[4] 'Du Scotome Scintillant,' 1875.

facts and considerations that hemiopia may be caused either by lesion of the optic tract, or from cortical or subcortical lesions in the occipito-angular region, it becomes an important question whether we can distinguish during life between the one and the other.

On this point we are naturally considerably aided by the simultaneous occurrence of symptoms indicative of a cerebral origin, such as hemiplegia, hemianæsthesia, aphasia, &c., but these alone are not altogether sufficient, nor are they always present, nor would they exclude simultaneous implication of the optic tract by pressure or otherwise.

A perimetric investigation of the eyes in hemiopia, proved to be due to direct lesion of the optic tract[1] shows the visual field divided by an almost vertical line passing through the point of fixation. On the one side of this line vision is entirely abolished to the right or left, as the case may be, both centrally and peripherically. Each optic tract supplies precisely the corresponding half of both retinæ.

On the other hand, there are many cases of homonymous lateral hemiopia, in which, though the lateral defect has been of indefinite duration, central vision is retained in both eyes for some distance on all sides of the point of fixation.

This is a fact of great importance and signification, and one to which I would direct especial attention as likely to furnish a means of diagnosis between cerebral and peripheral hemiopia.

In cases where central vision is retained for some degrees on all sides of the point of fixation I should regard the cause of the hemiopia as cerebral. The facts above recorded show that the angular gyrus of each hemisphere has a bilateral relation with the centre of the retina, so that, notwithstanding the complete destruction of the visual centre in one hemisphere, central vision is possible with both eyes, through the sound angular gyrus. Hemiopia therefore may exist towards the side opposite the lesion, with retention of central vision in both eyes.

The facts both of experiment and of cerebral hemianæsthesia, indicate that the area of central vision is more exten-

[1] Gowers' case, supra cit.

sive on the eye opposite the sound side; but central vision, more or less, is still possible though the opposite centre has been destroyed.

A rigid perimetric investigation is therefore a matter of great importance in reference to this question, and a careful comparison of the relative extent of the area of central vision in each eye desirable.

In many of the cases of hemiopia supposed to be due to injury or disease of the occipito-angular region, the perimetric investigation has not been recorded in such a way as to settle this point.

But in the case of Patrick Hughes,[1] Drs. Keen and Thomson found that in the right eye—the hemiopic side—"at the point of fixation the vertical line which bisected the field seemed to deviate slightly towards the defective side."[2] The figure they give shows a distinct area of central vision, interrupting the vertical line above and below the point of fixation.

Though they suggest excentric fixation and the possibility of the patient having followed the light inadvertently, yet the facts are more in accordance with the explanation I have suggested, viz. the functional activity of the sound angular gyrus.

Hirschberg regards it as the rule that in homonymous hemiopia the vertical line always diverges towards the defective side near the point of fixation, so as to have an area of central vision ranging from 3° to 5°. Where this is so, we may regard the hemiopia as of cerebral origin. And I am of opinion that careful examination will in all cases of cerebral hemiopia establish a greater clearness of central vision and over a larger area, in the eye on the side of lesion than in the opposite. An examination of some of the cases with the perimeters given by Schweigger,[3] also bears this out. The point, however, is one which I must leave to ophthalmologists.

It is a question whether there is any difference between cerebral and peripheral hemiopia, in respect to the degree and time of occurrence of atrophic changes in the optic disc.

[1] Op. cit. p. 126.

[2] 'Archiv f. Ophthal.,' Bd. xxii. Abtheil. 3, 1875.

[3] See page 470.

The statements as to the occurrence of atrophy in hemiopia are somewhat at variance. In many cases, even of long duration, ophthalmoscopic examination seems to have been negative. Gowers[1] has uniformly failed to discover it. Others again have described it, but while some have seen atrophic changes in both papillæ; others again, as Mauthner, state that in homonymous hemiopia atrophic changes are visible only in the disc on the hemiopic side. In a case of right hemiopia, however, of over ten years duration, which I have carefully examined in reference to this point, I have found atrophy of both discs, but certainly more pronounced on the right.

It is certain, however, that atrophy of the optic nerve does not occur so readily as atrophy in cases of lesion of peripheral nerves in general, a fact which may be due, as Gowers suggests, to the so-called trophic influence of the retina itself.

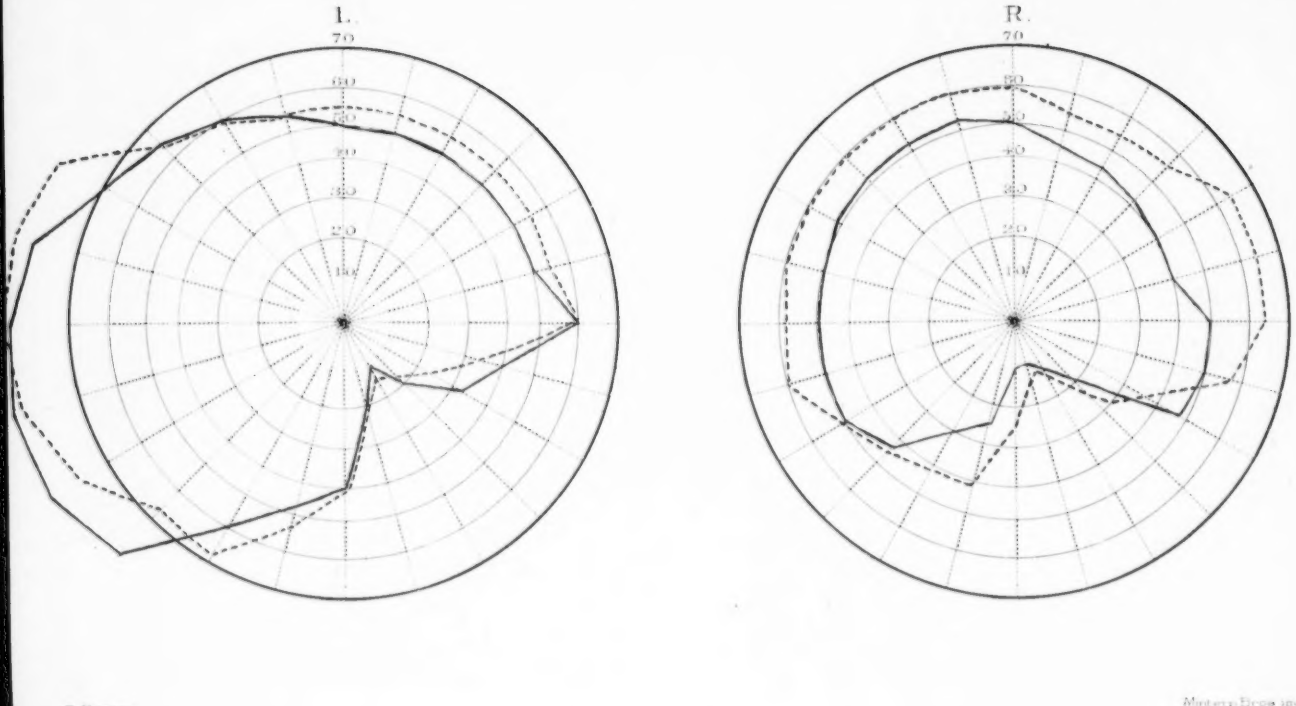
And this is supported by the fact that also in heteronymous, temporal or nasal, hemiopia, depending without doubt on peripheral lesion, the ophthalmoscopic examination is often, if not constantly, negative.

The question is one, however, which still requires investigation. That pale atrophy of the optic discs may occur from purely cerebral lesions altogether apart from lesion of the optic tracts, is a fact which the experiments of Dr. Yeo and myself—to be published in detail at a future date—clearly indicate.

The mere fact of the occurrence of optic atrophy would not therefore appear to constitute a means of diagnosing cerebral from peripheral hemiopia, but we might say that a rapidly occurring and decided atrophy of the optic discs would indicate peripheral rather than cerebral hemiopia, as direct lesions of the optic tract are more likely to be propagated to the retinal expansion of the optic nerve.

As to the duration of hemiopia depending on cerebral lesion, if we might argue without qualification from the monkey to man, we might expect that it should in time clear up and disappear. But the facts relating to cerebral amblyopia in connection with lesion of the posterior segment of the internal capsule seem to show that the duration of visual

[1] 'Brit. Med. Jour.' Nov. 1877.



C. Bergeat.

Mintern Bros. imp.

defects in man is much greater than in the monkey. Persistent defect in the acuity of visual perception and discrimination can be demonstrated in man, long after any defect as to mere light and shade, on which we have mainly to rely, can be recognised in the monkey's behaviour and modes of activity. It is further quite possible that the recovery from hemiopia in this animal may not amount to fine visual discrimination, though the perception of light may be sufficiently restored for ordinary modes of activity. The higher specialisation of the visual sense and important intellectual relations of binocular vision in man would lead us to believe that recovery after total destruction of the visual centres in one hemisphere would rarely if ever be absolutely perfect. The question is whether recovery to some degree at least may occur. The prognosis in cerebral hemiopia is generally held to be unfavourable. The hemiopic defect existed in Keen and Thomson's case (p. 470) eight years after the infliction of the injury to the brain.

In a case recently under my observation, however, of which the following is a condensed abstract of the notes, the symptoms as to vision were such as indicate gradual recovery from an extensive damage of the visual centres, and perhaps may throw light on certain forms of scotomata sometimes met with, the pathology of which is still obscure.

Case III.—J. S., ætat. 43, admitted into the National Hospital for Paralysis and Epilepsy on March 5th, 1880. Had an apoplectic seizure, with more or less unconsciousness for several days in Aug. 1879. On regaining consciousness, found that he was paralysed as to sensation and motion on the right side, with some degree of aphasia. He observed at this time that he could only see the left half of objects.

At the time of admission he was still suffering from considerable right hemiparesis and right hemianæsthesia, both as to tactile and muscular sensibility, hearing, and taste. Smell, however, was abolished in the left nostril instead of the right as usual.

The note of the examination of the eyes simple states that right hemiopia existed, but unfortunately no exact perimetric examination was made at this date.

The perimeter taken on 23rd March (Plate III.) shows a

want of symmetry in the visual fields, the right being considerably smaller than the left. At the same time there is in both a symmetrical quadrant-shaped scotoma downwards and to the right. The areas of colour-vision were found to correspond to the normal. On May 21, after two months' treatment, considerable improvement had occurred both as to motion and sensation on the right side.

The perimeter at this date shows that the visual field has become enlarged correspondingly in both eyes, the want of symmetry being retained.

The scotoma downwards and to the right still continues.

The ophthalmic appearances were negative.

The other symptoms in this case and the want of symmetry in the visual fields all indicate a cerebral origin. If we suppose, though unfortunately it cannot be demonstrated, that there was true homonymous lateral hemiopia in this case—and the patient was very positive as not having been able to see to the right either upwards or downwards at first—this might be looked upon as a partial recovery from cerebral hemiopia, and defects of this kind may be regarded as the remnants of a hemiopia at first complete. I find that a similar case in which complete recovery ensued has been recorded by Lang.[1] But the explanation of this and similar cases which most readily occurs is that the cerebral lesion has been only partial, and that, as in aphasia, the recovery is not so much the compensatory activity of the centres in the other hemisphere as the restoration, more or less complete, of the centres which have been damaged.

I have not been able to find any cases on record of hemiopia undoubtedly of cerebral origin in which the centres have been proved to be completely destroyed in one hemisphere, followed by recovery, unless we can regard the cases of latent occipital lesions as such. On referring to the recently published lectures by Mauthner,[2] which I have seen just before sending these pages to press, I find that he regards the prognosis of true homonymous hemiopia as almost invariably unfavourable, and that if it has existed for a few weeks a

[1] 'Centralbl. f. pr. Augenheilkunde,' July 1880.

[2] 'Vorträge aus dem Gesammtgeb. der Augenheilkunde,' Heft. 6–8. 1881.

restitutio ad integrum is not to be looked for as at all probable (p. 371).

He however records a case of right hemiopia (p. 365) of thirteen years' duration at the time of the first examination, which four years subsequently, i.e. therefore seventeen years after its occurrence, had recovered so far that on the right half of the fields formerly quite dark, not merely was light perceived, but a considerable amount of visual perception had returned. Colour perception remained quite abolished, however.

It is doubtful whether this was a case of true cerebral hemiopia, however, as visual defect in both fields was said to have reached quite up to the fixation point.

But it is a point worthy of future investigation, even in long-standing and apparently incurable hemiopia of cerebral origin, whether indications may not be discoverable of gradual improvement by the compensatory action of the sound visual centres, and whether this may not be fostered and stimulated.

DESCRIPTION OF PLATES.

PLATE I.—Field of Vision, R(ight) and L(eft), of Thomas H. (Case I., p. 459.)
The continuous line indicates the extent of the field of vision on April 24.
The dotted " " " " June 5.

PLATE II.—Field of Vision, Right and Left, of Hannah O. (Case II., p. 461.)
The continuous line indicates the extent of the field of vision on June 11.
The dotted " " " " July 30.

PLATE III.—Field of Vision, Right and Left, of James S. (Case III., p. 475.)
The continuous line indicates the extent of the field of vision on March 23.
The dotted " " " " May 21.

VISIBLE MUSCULAR CONDITIONS AS EXPRESSIVE OF STATES OF THE BRAIN AND NERVE CENTRES.

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COARSE or extensive paralyses such as hemiplegia, paraplegia and other profound disturbances of the muscular system have received much attention from clinical and pathological observers, and by the accumulation of their joint observations much knowledge has been gained as to the symptoms that result from lesion of certain portions of the brain. This should encourage us to observe in all cases the conditions of the muscles, knowing that the movements correspond to the states of certain nerve-centres, and looking upon such nerve-muscular conditions as indications or expressions of the states of those centres.

Epilepsies has been closely observed and accurately described, with the result of adding to our knowledge of brain conditions. Chorea, paralysis agitans, and the movements seen in athetosis, and disseminated sclerosis of the cord, were described as nerve-muscular conditions, and were empirically observed before their pathology was discovered. In these cases by studying the palsies, spasms, tremors, rhythmical and other forms of muscular action, we are enabled to gain some idea of the state of different portions of the brain and spinal cord. All expression of feeling is effected by muscular action, whether it be by words, by facial movement or gesture, movements effected by voluntary muscles; or expression may be produced by dilatation of the pupil, erection of the hair,

or disturbed action of the heart, these being due to the conditions of inorganic muscular fibres.

For these reasons I have been accustomed to look rather closely at the nerve-muscular condition of "nervous cases" when seeking definite signs by which to describe them, sure of the axiom (with certain exceptions, such as conditions of muscular irritability, and muscular disease) that movements depend upon nerve-muscular stimulus originating in nerve-centres.

Examples may easily be given showing how we commonly judge of the state of the nervous system by muscular conditions. Note the stooping attitude and spiritless gait of a tired man as compared with that of the same individual when rested and refreshed. Incipient intoxication is indicated by a reeling gait, unsteady hand, and muscular tremor. Expression may be indicated by the position of the head, it is seen firmly upright in defiance, drooping in shame; it is commonly held on one side in nervous women, and girls convalescent from chorea, the first example cited of an asymmetrical gesture. The artist's brush or pencil, the sculptor's modelling tool and chisel, the pianist's and violinist's finger-touch, indicate the training and actual condition of the working of his brain. The educated and refined singer trains and refines his whole mind, i. e. his brain, and is well aware that his "whole soul" as he may express it, comes out in the action of the muscles concerned in producing his song, and musical notes.

In the infant the condition of the nervous system is best recorded in terms of nerve-muscular phenomena. It laughs and is playful; reflex action is well marked when a finger is placed in the child's hand, or mouth. The eyes are moved and directed towards any object looked at; these are conditions of healthy action. It is well known that in the convulsive state the fists are often closed with the thumbs turned in.

All these examples of expression are nerve-muscular conditions; the movement, the attitude, the gait result from states of the brain or spinal cord.

In the observations to be referred to, examples are chiefly drawn from the ocular and facial muscles, and those of the upper extremity. Muscular movements in the eyeball are

seen in the varying conditions of the pupil under the influence of light, and in the changes accompanying accommodation for near and distant vision.

In the iris we have a very sensitive and beautifully adjusted muscular mechanism, in connection with the third nerve of the brain and the sympathetic. The clinical study of this muscular organ and its indications were fully and carefully treated by Mr. Hutchinson in a paper in 'BRAIN,' Vol. I.

Movements produced by muscles outside the eye are seen in the act of directing the eyes in any given direction, these movements being so governed that the parallelism of the axes of the eyes is usually maintained; these co-ordinated movements being probably governed by some brain centre. In looking at objects about two inches from the face, these recti muscles outside the eyeball cause convergence of the axes of the eyeballs. To allow of such convergence, the simultaneous and equal contraction of the internal rectus muscle of each eye must be accompanied by the relaxation of the external recti; this complicated association of muscular conditions must be regulated by the nerve-centres. These movements are the normal.

We now pass on to consider departures from these normal conditions. I have shown that in deep anæsthesia from chloroform,[1] or in coma from alcoholism, or in the profound sleep of infants, the loss of associated movements of the eyes may be complete. If in an adult deeply under chloroform, the eyelids be gently raised, the pupils will be seen minutely contracted, often to a pin-point, the eyes having at the same time lost the parallelism of their axes. One eye may move upwards or outwards while the other remains quiet, or moves in a different direction, or at a different pace, thus causing a temporary and varying strabismus. Usually these movements are confined to the horizontal plane, less commonly the eyes assume a different level, one being in the horizontal plane, while the other is turned downwards. It is noteworthy that the average continuance of the eyes in the horizontal plane of the axes of the orbits, is in accordance with other examples

[1] See paper, 'Brit. Med. Journal,' March 10th, 1877.

of involuntary movements of the eyes, to be presently noted, e. g. nystagmus, and irregular jerking movements of the eyeballs. These movements I have frequently observed in the healthy subject, and seen that though they occurred thus irregularly while in coma, the action of the pupils and the co-ordination of the eye-movements was completely restored on recovering consciousness. In the profound sleep of an infant in its mother's arms the same loss of association of movements occurs, but at the moment of waking the pupils dilate, and co-ordination of movements is restored; the child must be profoundly asleep to allow of the eyelids being raised without awaking it.

Case I.—At the Children's Hospital, Birmingham, in 1876, I saw a girl three years of age the subject of permanent hemiplegic paralysis, following convulsions in infancy. She had never spoken, was constantly dribbling, and idiotic in manner. There was an occasional loss of the parallelism of the eyes; one would remain at rest, while the other wandered inwards or outwards; this was a chronic condition in the child, who was suffering from no acute disease. The symptom appeared due to the defective condition of the brain.

Case II.—Through the kindness of my friend Dr. Fletcher Beach, I had the opportunity in 1877 of seeing in the Clapton Asylum a microcephalic idiot of very low development, in whom the eyes almost habitually wandered about independently of one another. In very weakly infants, not the known subjects of brain disease, the loss of associated movements may be very distinctly seen at times while awake and sucking at a bottle. In cases of meningitis, and other conditions of coarse brain disease, the same condition is sometimes seen.

Lastly, it is interesting to call to mind that in many of the lower animals there is the power of moving either eye separately and independently, so that that centre which co-ordinates the movements of the two eyes may be looked upon as a "later centre," i. e. one more recently developed in the ascent of Man. Probably some interesting and important points concerning the movements of the eyes might be elucidated by considering them in connection with those of the lower vertebrata.

Nystagmus is a condition producing a rapid oscillation of the eyeballs; usually the parallelism of the axes of the eyes is maintained, and the movements are in most cases in the horizontal plane of the axes of the orbits. Occasionally the movements are vertical, as in the cases of two brothers with retinitis pigmentosa, frequently seen in the clinique at Moorfields about 1870.

These cases of vertical nystagmus are uncommon, so also I believe, it is unusual to have one eye only thus affected.

Case III.—Emily B., aged 4 years, was sister to an infant under my care at the East London Children's Hospital for congenital syphilis. When seen, nystagmus of the right eye was at once observed. There was inconstant vertical oscillation of the right eyeball, without any lateral movement; the movement was seen when the child looked at distant objects, not when looking at near objects; there was no nystagmus of the left eye. The pupils were equal; there was no strabismus. The normal movement of the eyes could be performed, together with the natural convergence for near vision. Ophthalmoscopic examination was impossible, on account of photophobia. There was good vision in both eyes; the child was intelligent.

Case IV.—In an infant which I had the opportunity of seeing at the London Hospital while she was under the care of Mr. Hutchinson, there was congenital nystagmus of both eyes; the rapidity of movement in the two eyes varied greatly, but it was impossible to count the oscillations. This would appear to indicate that the nerve-centres ruling either eye and regulating the tension of one rectus muscle when its opponent is relaxed, act more independently than in the normally developed brain, i. e. the centre whose office it is to co-ordinate the associated movements of the two eyeballs is feeble or absent in such a case, as it appears absent in some idiots, and paralysed (in healthy brains) in deep coma.

Case V.—William D. was the subject of right hemiplegia and presented syphilitic lesions in the soft palate, pharynx and bones, and there was atrophy of the right half of the tongue.

There was no strabismus, and he could move the eyes in any direction; when the eyes were directed in an extreme

degree to either side horizontal oscillatory movements resulted, the lateral jerks reminding one of the more rapid oscillations of nystagmus, but these movements only occurred when the eyes were directed towards the right or left hand; no such oscillations resulted on looking upwards or downwards. Another noteworthy phenomenon concerning the muscles of the eyes is the fact of convergence for near vision: an additional example of movements in the horizontal plane of the axis of the orbits. This movement is well seen in a healthy individual by causing him to look at a distant object and observing the size of his pupils and the parallelism of his eyes, then causing him to look at his nose the eyes are seen to converge, directing their axes towards the object, at the same time the pupils contract.

Case VI.—Mr. Priestley Smith, in the Ophthalmic Hospital Reports, vol. viii. part ii. narrates the case of a lad in whom the lateral movements of the eyes were impaired. "Both eyes were fixedly turned towards the patient's right. The deviation, measured by prisms, equalled twenty degrees in each when looking at a distance. The head was kept turned towards the left to counteract the position of the eyes. An object was moved from right to left, and left to right in front of the patient. Neither eye could follow it to the smallest extent towards the left, but both, on the contrary, were capable of a very slight additional turning towards the right. This they performed, however, in a jerking manner, and at once returned to their former position. An object was then held at ten feet distance and rather towards the patient's right, so that his eyes could be fixed on it, and it was then made to approach his face. Both eyes steadily converged so as to remain fixed on the object until it was only five inches from the face. This involved a remarkable phenomenon, viz. the right eye, which had before refused to move towards the left, now made a considerable excursion (about fifteen degrees) in that direction." In this highly interesting case collateral deviation of the eyes was marked, the power of convergence for near objects being retained.

Ferrier has shown[1] that there exists in the convolutions of

[1] 'Functions of the Brain,' p. 229.

the frontal region a brain centre, which when excited causes both eyes to turn away from the side excited; if on the contrary this centre be destroyed instead of excited, the corresponding centre in the other half of the brain, acting unopposed, turns both eyes towards the side of lesion. Such a lateral deviation of the eyes, parallelism of their axes being maintained, is a common condition in cases of hemispasm and in recent hemiplegia.

In health the greater number of ordinary movements of the eyes are probably in the horizontal plane of the axes of the orbits. Thus the movements of accommodation for near vision are in this plane, in nystagmus the oscillations are usually so, also the jerking movements in the case V., and the involuntary movements with loss of parallelism seen in coma, are mostly horizontal. The facts point to the horizontal movements as the commonest, the least voluntary, and probably the least intellectual. The horizontal movements of the eyeballs do not involve movements of the eyelids by muscular action, i. e., the horizontal movements of the eyeballs do not cause simultaneous movements of the muscles of the eyelids, therefore I think horizontal movements the least intellectual. To turn to vertical movements. Dr. Gowers has shown* that the movements of the lower eyelid constitute a simpler problem than those of the upper lid. The lower lid follows the movements of the eyeball upwards and downwards, but not very closely. No muscular mechanism exists which can cause the downward movements of the lower lid; such movement is produced by the movement of the eyeball acting mechanically upon the lower lid. The upper lid possesses a more complex mechanism. The descent of the lower lid on downward rotation of the globe is not due to the contraction of the orbicularis. Simultaneous with the descent of the upper lid in the downward movement of the eyeball, there must be a relaxation of the levator. In upward rotation of the eyeball, contraction of the levator, is associated with that of the superior rectus. The association of the levator and superior rectus suggests that both are relaxed or energised in similar degree when the eyeball is moved upwards or downwards; in

* 'Med. Chir. Trans.' vols. i. and ii.

accordance with the law, which it is evident must obtain in all muscular actions, that the opponents of the muscles producing the movement, are relaxed in exact proportion to the degree of movement produced. Thus in the upward vertical movements of the eyeballs, a more complex movement and especial muscles come into play.

Contrasting the horizontal with the vertical movements, we see that the former involve only movements of the rcecti muscles. Of movements of the eyeball in the vertical plane those in the downward direction involve contraction of the inferior rectus only, but at the same time the levator must be relaxed; this co-existent relaxation of the levator and contraction of the inferior rectus suggest that the downward movements are more intellectual than the horizontal. In upward movements the levator contracts synergically with the superior rectus, and the co-existent activity of the two muscles points to these as probably the most intellectual of all. This is again another point as to which it would be exceedingly interesting to know the teachings of comparative anatomy to see whether this power of upward turning of the eyeball, with elevation of the upper lid, be not a later developed power.

From direct observation it appears to me that intellectuality is represented by the movements of the eyeballs in their orbits. When an individual in looking at an object moves the eyes by the action of the recti muscles so as to direct them towards it, the movement is more intellectual than when the head is turned so as to direct the eyes in the required direction. A bright, healthy well-developed infant turns its eyes well in the orbits in looking about, not so a dull wasted child.

Again, intellectual people usually move the eyes in their orbits in looking at any object; the low and vulgar often move the head in the direction required. Here again is a question as to the meaning of which comparative anatomy might give some light.

In chorea we frequently see that although the eyes never lose their parallelism, they are often jerked about in a perfectly involuntary manner, and clearly without the patient desiring to look at the successive objects towards which the eyes are turned. It would appear that the brain-centre which governs

the co-ordinated movements of the eyes is never paralysed in chorea as in conditions of coma, but that it may be "choreic"; this centre then may be temporarily paralysed in healthy infants, or in healthy adults by chloroform; it may be absent in idiots; in weakly infants the slight disturbance of sucking (cerebral anæmia?) may paralyse for the time this centre; this centre is absent in many lower vertebrata.

In making observations on these associated movements of the eyes we are strongly reminded of Ferrier's experiments* on excitation of different portions of the cerebellum. I quote a few results. "Both eyes turn to the left or right in a horizontal plane"; "both eyes move straight downwards"; "both eyes move upwards and to the left"; "both eyes move upwards," etc. These results of experiments suggest that possibly it is the condition of these cerebellar centres that leads to the eye movements described and referred to.

We now come to the study of visible conditions of the facial muscles in expression of the state of the nerve-centres.

The method I have used in analysing the muscular condition of faces is as follows:—holding a piece of paper with one edge vertical, either half of the face is covered in turn, it is thus seen whether the face is symmetrical. Again the face may be divided into three zones by holding the paper with one margin horizontal, leaving the forehead above the eyebrows uncovered; or the face below the lower margin of the orbit may alone be exposed, showing the mouth, most of the cheeks, and the alæ nasi; or again, the middle zone including the eyelids may be viewed alone.

Most of the more definite forms of expression by muscular action are symmetrical, and the face is no exception to this rule. Asymmetry of the face is not met with in many instances. It is seen in facial palsy, whether from disease of the portio dura of the seventh nerve, or from brain disease; it is also seen in the expression called sneering when a one-sided muscular action partially uncovers the canine tooth; and in winking. Occasionally in very nervous people a one-sided grimace may be seen. Other cases of asymmetry of the face may be seen, but are rare.

[1] Op. cit. p. 98.

Before proceeding to describe symmetrical conditions of the face illustrative of the subject under consideration, it may be well to consider what we see when we look at a face.

1. There is its *form*, outline, and the relative position of parts.

2. *Colour*, a condition of the surface which may be in part due to pigmentation of the skin and other conditions, as well as to the state of its circulation.

3. *Movement* of the parts of the face from tone, action, or relaxation of the muscles. It is with these conditions of the muscles that we are principally occupied.

Certain commonly marked lines in the face exist as the result of the puckerings produced by muscular action. In the frontal zone horizontal lines are produced by the action of the occipito-frontalis, and vertical lines are produced by the action of the corrugators. In the middle zone the most pronounced lines are the naso-labial grooves; we also specially here observe the lines of the upper and lower lips, and the positions of the angles of the mouth. The mouth may be widened, or one or both angles may be drawn upwards and outwards or downwards.

The attempt has been made to determine something of the intellectuality of the different facial muscles. The problem was commenced on the negative side by observing the conditions of the face in fifteen idiots. In conjunction with Dr. Fletcher Beach, of Darenth Asylum, I analysed each face according to the following form :—

General muscular condition . .	The action, or relaxation, of the muscles of the limbs and body generally, were noted.
Face	Facial aspect, and muscles in action or relaxation.
Upper zone . .	Frontal region, occipito-frontalis and corrugator.
Middle zone . .	Eyelids and orbicularis oculi.
Lower zone . . .	Mouth muscles; muscles of nose; cheeks.

Summarising from such forms the muscles most often seen in meaningless action, the following results are obtained :—

Occipito-frontalis	11	Depressor anguli oris . .	5
Zygomas	8	Orbicularis oculi . . .	3
Corrugator supercilii . . .	7	Grief-muscle	2

With regard to the teaching of these figures, so far as these few cases go, it shows the frequency with which these muscles

respectively come into spontaneous action in a meaningless manner. This is perhaps some indication of the degree of their intellectual representation. Thus the grief-muscle and orbicularis oculi were much less frequently thrown into meaningless action than the occipito-frontalis and zygomatic, and probably these former are much more expressive of intellectuality than these latter.

In the above table the number 7 is put against the Corrugator; but this I think needs explanation; for from direct observation in healthy intellectual faces, I think that this muscle is one of the most intellectual. If, instead of putting one against the name of a muscle as it may be seen frequently in action in a particular face, we put one or one half, according as its action is marked or only slightly indicated, on summarising these numbers we get for the occipito-frontalis 10·5, for the corrugator 5·0. This result indicates the slight action of the corrugators in the faces of idiots as compared with the occipito-frontalis.

Again, applying direct observation to the other side of the question, and noting which muscles are most frequently put in action in the faces of intellectual people in the expression of their mental states, I think that we see intellectuality most commonly expressed in the frontal and middle zones, and by the action of the corrugator and orbicularis oculi muscles.

Among out-patients it has frequently been possible from the muscular condition of the frontal region (principally increased tone or contraction of the corrugators), and depression of the angles of the mouth (dep. ang. oris) to accept the facial appearance as a physical sign of the "mental state" of intellectual suffering, and physical or organic suffering. On inquiry in such marked cases some painful condition and some source of anxiety or mental distress has almost invariably been found.

Much has been said[1] at various times about facial expression. I will not add thereto, but suggest that to describe such expressions in anatomical terms is a matter of physiological importance, as giving more exact indications of the nerve-centres.

[1] See Darwin on Expression of the Emotions; Bell on the Face; Camper, 1780; Bulwer, 1649.

Taking the face as a clinical region of observation, special points sometimes observable in the different zones may be described. When specially studying the faces of patients, the subjects of recurrent headaches, and analysing them daily, my attention was particularly drawn to the middle zone.

It is not uncommon to observe that an individual "looks as if he had a headache." Analysing such faces it soon became noticeable that there was a look of depression, heaviness, fulness about the eyes, especially about the under eyelid. It appeared that this expression must be due principally to the condition of the orbicularis palpebrarum. There was obviously no pitting on pressure, no œdema, and when the face is œdematous this relaxed look is not seen. Specially observing the orbicular muscle and the parts adjacent, there seemed to be a loss of tone in it; there was an appearance of fulness and flabbiness; the skin hung too loosely, with an increase in the number of folds; and, in place of falling against the lower eyelid neatly, as a convex surface, it fell more or less in a plane from the ciliary margin to the lower margin of the orbit. This condition is often seen best by looking at the patient's face in profile. It was often seen when the skin was healthy and elastic; and that it is due to the muscle seems demonstrated by the change produced if the patient can be induced to laugh, when the muscle is energised, recovers its tone, tucks in the skin well against the eyeball, and the expression of headache is lost for a time. It is not suggested that this muscular condition indicates only the states producing headache; it appears common in after-states of depression.

It may be remarked that the eyeball is probably almost entirely lacking in expression. If a man wear a mask, showing the eyes only, there is no certain expression. It is the custom in some parts of Italy for men to beg in silence, wearing a loose garb, and hood covering the face, with holes showing the eyeballs only, and the absence of expression is marked.

Doubtless in rage we have the dilated pupil, and it contracts with accommodation for near vision; it may also have a characteristic pigmentation; but, speaking generally, the eyeball almost lacks expression, and the special appearances observed about the eye are due to conditions of the eyelids

and the muscles which move them (the orbicularis and levator palpebræ), and in some degree to the muscles which move the eyeballs.

The orbicularis oculi is not solely supplied by the facial nerve, but has also some supply of nerves from the sympathetic.

It is likewise one of the muscles not greatly paralysed by facial palsy due to brain disease. Indeed in Bell's paralysis the eye can generally be closed.

Case VII.—In a case of congenital Bell's paralysis,[1] from ear disease, in an infant, accompanying hemiplegia on the opposite side, the eyelids on the side of paralysis were closed when the face was blown on. This muscle is much less paralysed than the zygomatic in cerebral facial palsy.

In the lower zone are many points of interest to be noted. With regard to the depression of the angles of the mouth in feelings of sorrow, I have frequently observed that when a mother has just lost her child there is a marked depression as from physical suffering.

Case VIII.—In the case of a mother speaking to me of the loss of her children four years previously, there was no depression of the angles of the mouth, but contraction of the corrugators; this would seem to point to the passing away in the course of years of the expression of mere physical suffering from the loss of her children, and the more permanent expression of intellectual suffering from the bereavement.

Case IX.—A woman came to see me as an out-patient, ill with the effects of distress at the loss of a son, aged 21 years, and married only three months. He had died of small-pox a month previously. Her aspect was emotional, she flushed much, and there was marked expression of distress, indicated principally in the frontal region by action of the corrugators, and slight contraction of the inner portion of the occipito-frontalis. The angles of the mouth were not depressed. The face appeared to indicate mental distress and anxiety rather than mental suffering.

The compressoris alæ nasi under ordinary circumstances and in dyspnœa act rhythmically, and synchronously with the respiratory movements, under the guidance of the respiratory

[1] 'Brain,' Part VIII.

centre. In slowly advancing death the alæ nasi may become paralysed, and so collapse on inspiration. Again, in chorea these muscles are often distinctly choreic, being jerked with the other facial muscles, thus seeming to indicate they receive the choreic impulse from the facial centre, not from the respiratory, as they do not jerk synchronously with the other respiratory muscles.

Among asymmetrical conditions of the face the most marked are due to paralysis. Facial paralysis occurs from two principal causes—(1) lesion of the facial nerve; and (2) lesion of one side of the brain, attended with paralysis of the upper and lower extremity, and of the face on the opposite side. In the case of facial palsy due to brain disease, the muscles about the angle of the mouth are principally weakened. This is illustrated by the photograph of a man the subject of left hemiplegia. If either vertical half of the face be covered, it is evident we have an asymmetrical condition. If then we cover each zone in succession, and again look at each zone separately, we find the asymmetry is in the lower zone about the mouth; it consists in a falling of the left angle of the mouth from weakness of the muscles, which expand the mouth and draw its angle upwards and outwards.

This may be compared with the photograph of a case of Bell's paralysis on the right side, in which all the facial zones are affected in an asymmetrical manner, the muscles all being weakened on the same side.

In connection with the fact that brain disease causing one-sided facial paralysis affects the muscles about the angle of the mouth the most, we are reminded that the zygomatics are among the most frequently active muscles in the movements of the faces of idiots, as recorded in the table. These muscles are also sometimes seen acting very asymmetrically in very nervous people. A very moderate amount of brain disturbance appears to cause irregular action or weakening of these muscles.

Again we are reminded of Ferrier's[1] experiments. I quote the results of the excitation of points in the ascending frontal convolution: "(7) action of the zygomatics, by which the angle of the mouth is retracted and elevated." (8) "Elevation

[1] Op. cit. p. 143.

of the ala of the nose and upper lip, with depression of the lower lip, so as to expose the canine tooth on the opposite side."

Some points in favour of the clinical study of nerve-muscular conditions of the face are illustrated by the passive, expressionless face which may be woke up, "lighted up," made to express "the whole soul in the face" simply by conditions of tension of the facial muscles resulting from the mental state. Thus often great and most pleasing beauty is seen in faces unattractive when at rest. Conversely some faces are beautiful in their passive condition, but lack expression and interest when in action from mental work; such women talk but little. Surely from these two considerations it is suggested that the passive form and colour of a face are qualities not as great, not as much mind-indicating, as the mobile expressions produced by muscular tension.

Possibly we may for a moment be allowed to step out of our groove and see how education, and thoughts and habits of thought, and feeling, implanted in the individual can and do produce an effect on the individual's higher nerve-centres, for certainly direct observation shows that these principles, these forces or modes of force alter the facial expression.

Other conditions of the face expressing emotion have been described and illustrated by Darwin. If these expressions be due to muscular action, they must be due to the condition of the nerve-centres; and it is as indicating their conditions that they are of special interest in the pursuit of our special subject. The physiologist and practical physician are principally concerned with the human body as it now exists, and to them it is not a case of the emotion (feeling), causing expression, or the mind showing itself in the face, but rather a condition of the muscles of the face caused by the condition of nerve-centres, and indicating this condition.

Now, passing on to observations of the muscles of the upper extremity. Probably the most expressive muscles are those which move the fingers, i. e. those which produce the finer movements. These finer movements appear more to represent the brain conditions than do the coarser movements, e.g. those of the shoulder or elbow.

It is of course in the free or disengaged hand that we must look for examples illustrating the condition of the brain which governs it. If the muscles be employed in some definite act, such as holding an object, or in an act of manipulation such as sewing, then the movements are directed to accomplish the aim attempted, and are not simply indicative of the condition of the brain, as may be the case with the free hand when unconsciously expressing the mental condition by gesticulation. When, on the contrary, the hands are left free and disengaged, as the hand of the orator which unconsciously expresses by its position or movement the general mental state of the speaker, we have in this muscular movement an expression of the man's mind. It is as reasonable to look for the state of the mind to be expressed in the position and action of the hand engaged in definite voluntary, purposive acts, as to look in the face when the sun is shining full in the eyes, or the lips are engaged in eating, or moved with the other movements of dyspnœa. Still it is true that in either case the manner of performing the act may be indicative of the mental state, but the muscles of the face or hand are not engaged in expressing the mental state.

In Art at the present day we but seldom see the hands represented as disengaged; usually they are painted or sculptured holding some object, or resting on some part of the figure; such are hands engaged or resting from labour, or performing some act of toil, not engaged in expressing the action of the mind.[1]

I have spoken of the finer movements of the hand as the most expressive of the condition of the brain. Movements are spoken of as coarse when performed by large muscles, e.g., movements of the shoulder and hip-joint are coarser than those of the wrist and ankle. Fine movements are performed by smaller muscles, and are confined to small arcs, e.g., slight movements of the fingers; the smaller the arc through which the finger moves, the finer is its movement said to be.

The finer movements of the hand have been spoken of as most expressive of the condition of the brain; some reasons for this statement may now be given. 1. In hemiplegia from

[1] Examples of the disengaged hand are seen in the statues of Cain in the Pitti, Florence; the Venus de' Medici; and the Diana, British Museum.

brain disease the finger movements are most damaged, and the latest to recover. 2. Finger movements are much more damaged by brain disease than by spinal disease.

It may be convenient to commence this study by the consideration of four principal positions of the hand, due to, and representative of the condition of nerve-centres. These may be easily described, and perhaps will be useful in describing conditions of departure therefrom.

In a former paper on "Recurrent Headaches in Children" I described the position of the "nervous hand."[1]

When a very nervous child, or one convalescent from chorea, holds out its hands in front, on a level with the shoulder, and with the fingers spread out, we commonly see this nervous hand. As the palms are turned down, the wrist droops slightly. The metacarpo-phalangeal joints are moderately extended, the first and second internodes being either flexed or kept straight. The thumb is simply extended backwards, and somewhat abducted from the fingers. This position is also maintained when the palms are turned upwards. In such cases there is very commonly finger twitching, of which notice will be taken further on.

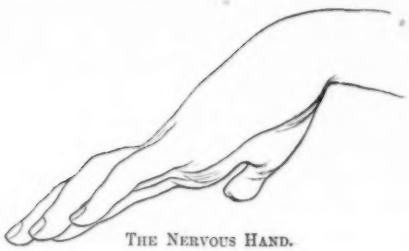
With regard to indications of states of the nerve-centres by states of the muscles, there appears to be evidence[2] that in opposite states of the nerve-centres the respective conditions of the muscles are in direct antithesis. Probably also the converse is true, that opposite conditions of the muscles indicate opposite conditions of the nerve-centres. Let us look at the antithesis of the "nervous hand." In applying this principle of antithesis, we must reverse each relative position of the phalanges and joints.

	The Nervous Hand	*versus*	The Energetic Hand.
Wrist	flexed.		extended.
Metacarpo-phalangeals . .	extended		flexed.
1st internodes	flexed		extended.
2nd internodes	flexed or ortho-extended		extended.
Thumb, metacarpo-phalangeal	extended		flexed.
„ 1st internode . . .	extended		flexed.
Phalanges relative position .	slight abduction . . .		abducted.

Now we may inquire if this antithesis of the nervous hand be indicative of a condition of the nerve-centres the opposite

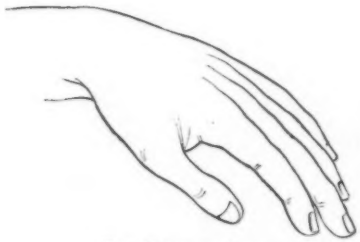
[1] 'Brain,' October 1880.

[2] See Darwin.



THE NERVOUS HAND.

THE ENERGETIC HAND.



THE HAND AT REST.

THE STRAIGHT HAND.

NOTE.—The hand should be represented as perfectly straight with the forearm.

To face p. 494.

of the "nervous condition." This position is represented in the Diana in the British Museum, and well corresponds with the general forcible attitude of the figure. In one point, however, this hand of the Diana differs from the exact antithesis of the nervous hand, in that the first and second internodes are flexed. The forcible character of this position of a free or disengaged hand is well seen in contrast with the nervous hand of the adjacent Venus; and it was in comparing these that I saw the one to be the antithesis of the other.

This energetic or powerful hand is probably in life seen only under certain mental conditions, or as it is preferable to say in certain conditions of the brain producing what we call mental states, such states of the mind giving energy and the feeling of strength. I do not know that it is to be seen as the result of any pathological condition of the brain. It is, I think, seen in forcible expressions of feeling; and in astonishment, as expressing which it is figured by Darwin.

The most natural position of the hand is doubtless that of rest, and here analysis shows all the joints in the position of flexion. The hand may be seen in perfect rest during sleep, or when the man is resting, engaged in quiet unexciting conversation. The following is the analysis :—

THE HAND IN REST	*versus*	HAND IN FRIGHT.
Wrist	flexed	extended.
Metacarpo-phalangeals. . .	flexed	ortho-extended.
1st internodes	flexed	ortho-extended.
2nd internodes	flexed	ortho-extended.
Thumb, metacarpo-phalangeal	flexed	ortho-extended.
„ 1st internode . . .	flexed	extended.
Phalanges, relative position .	abducted	abducted.

This analysis represents a hand in complete flexion and its antithesis a hand in general extension or ortho-extension. The term ortho-extension is used to imply that the joint is so far extended as to place both the bones constituting the joint in the same straight line. If extension be full or complete, the bones form an obtuse angle with one another.

(To be continued.)

TETANUS.

BY SURGEON-MAJOR J. J. L. RATTON, M.D., MADRAS.

In a paper published in 'Brain,' Part VIII., January 1879, I endeavoured to show that peripheral nerve-irritation in some part of the body is invariably a factor in the causation of tetanus. That there are other factors at work, predisposing and exciting, "goes without saying," and to these points I will recur; but for the present I confine myself to peripheral nerve-irritation, believing that the subject requires further elucidation. There can be little doubt that the nerves implicated in tetanus are the ordinary motor or mixed nerves. This seems to have been proved, clinically and experimentally. The important question now arises, what is the nature of the irritation? Is it mechanical or tactile; pathological or painful; or a combination of both? Our limited knowledge of nerve-physiology and pathology would lead us to the conclusion that it must be either one or the other, or a mixture of the two. On the threshold of this investigation we are met by the difficulty of determining how external impressions are conveyed by the nerves. We know that the nerves are conductors. We know that they consist essentially of a semi-fluid conducting medium, contained in tubes, and strengthened by fibrous sheaths. We also know that it is not essential to the performance of a reflex act that the nerve implicated should conduct to the brain; but we do not know how, or what, the nerve conducts. We do not know whether nerve-force is one of the natural forces known to science, working through living cells, and thereby altered in its reactions; or whether it is a new and unknown quantity. We never shall know, probably, how a man lifts at will his little finger, without antecedent cause of any kind, except his own volition.

But this is a higher flight than necessary here. The question is, how are tactile sensations transmitted to the nerve-centres through the centripetal nerves?

I incline to the belief that tactile impressions are a form of motion; that as light and sound are appreciated by the eye, and ear, and brain, by their vibrations, so the appreciation of external objects, by the sense of touch, is a question of vibrations. And to this I have been led not only by analogy, but also by a very simple experiment, which illustrates the matter in a coarse exaggerated way. Any one who draws his finger over a rough surface, and then over a smooth one, notices at once the difference of vibrations communicated to the finger. It is by these vibrations he appreciates the one as rough, the other as smooth. And, *vice versâ*, if a person touch but lightly different objects, with his eyes blindfolded, he will be unable to give their most prominent physical characteristics until he has, by some little friction with the fingers, produced the corresponding vibrations. There is nothing very new in the proposition that vibrations, mechanically produced, by feeling with the fingers or by surface-contact with external bodies, are propagated, unchanged perhaps, through the diffluent centres of the nerve-tubes, to the nerve-centres, or to the brain, where they are appreciated and expended in the convolutions.

As a matter of fact, the waves of sound, with which we may compare the vibrations of objective feeling—though the latter are probably larger and more violent—travel at the rate of 4000 feet per second in water, and in solids with varying velocities, up to 16,000 feet per second. If we suppose tactile vibration transmitted at the lower rate, 4000 feet per second, through the semi-fluid matter of the nerves, a person six feet high would have cerebral consciousness of an object touching his foot in the 1–666th part of a second, quite fast enough to meet the facts of the case—the requirements of the hypothesis.

That the brain is partly formed to appreciate vibrations is probable from its convoluted shape, and is proved by the perception of light and sound. Here we have to deal with molecular vibrations, or waves, so delicate and ethereal, that special

organs, the eye and ear, have been developed for their reception. There can be little doubt that the waves of sound, caught upon the tympanum, are passed on to the brain, diminished in intensity it may be, but not otherwise changed. The science of acoustics is a monument of the appreciation of vibrations by ear, and eye, and brain. Heat and cold are also modes of motion. Moreover, heat, light, sound, and ordinary mechanical motion—I might even include electricity—are interchangeable modes of motion, and convertible one into another. I suppose that the molecular vibrations of heat and cold, different from others in wave-length, or rapidity, are conveyed to the brain by the nerves, acting as simple conductors, in the same way that ordinary tactile sensations are conveyed.

All this leads to the conclusion that man derives all his impression of external objects by vibrations, and that his nerves are, in one sense, merely conductors of vibrations, just as telegraph wires are conductors of electricity; though they have other, higher, and vital functions as well. It may be well to repeat here that these views are held only with reference to the centripetal nerves. How these vibrations react upon the nerve-centres, and what becomes of the nerves, and centres, when the individual perpetually bombarded by vibrations is asleep, I confess I do not know. This is a matter closely linked with the great question of how the will acts, with which I have no wish to meddle.

Before proceeding to apply the above hypothesis to the solution of the problem as to how peripheral nerve-irritation produces tetanus, let us see what pain is; for, as before stated, the irritation may be either tactile or painful, or both combined.

Pain, there is reason to believe, is an affection of the vaso-motor system of nerves. C. D. Radcliffe, M.D.,[1] looks upon pain as being the result of *irritation* of the vaso-motor nerves and contraction of the capillaries. He points out that in all diseases when inflammation, denoting *paralysis* of the vaso-motor nerves, begins, pain diminishes, except where fibrous coverings prevent swelling and cause tension. He

[1] Reynolds' 'System of Medicine,' vol. ii.

instances the pains in the cold stage of fever, and at the beginning of an attack of gout, before heat and swelling are developed, and other cases.

On the other hand, most men connect pain with inflammation and paralysis of the vaso-motor system. Heat, redness, pain, and swelling are the most prominent symptoms of inflammation; and it is hard to say in what order these symptoms appear, so closely do they occur together.

Lateral section of the spinal cord causes motor paralysis, with increased temperature and sensibility, on the side cut; loss of sensation with diminished temperature and no paralysis, on the opposite side. It would seem from this, Dr. Radcliffe remarks, that the injury had acted upon the vaso-motor nerves contained in the cord, as well as upon the common motor and sensory nerves, causing paralysis of the vasor-motor nerves on the side on which there is increased temperature and sensibility, and irritation of vaso-motor nerves on the side on which there is diminished temperature and anæsthesia. The value of this experiment depends upon the mode of distribution of the vaso-motor nerves in the cord. As they accompany the circulatory system, it is possible that they do not decussate, in which case the significance of the experiment would be completely altered. Be this as it may, pain has long been recognised as a disturbance of some kind of the vaso-motor or circulatory system, and even in its purely nervous forms, as the various neuralgias, excepting always hysteria, it is probably a form of inflammation.

Dr. S. Weir Mitchell, of Philadelphia ('BRAIN,' Part III.), chilled a limited portion of a mixed nerve until touch was lost; at this point the least tap on the nerve caused motion of all the muscles supplied, and some pain, showing that pain-sensation passed the frozen part, whilst tactile sensibility could not; they are therefore probably different things. His close study of therapeutic neurotomy in several cases leads him to ask, "but why should this operation, which annihilates touch, leave pain-power unaltered in the regions under the skin?" I infer it is because pain is an affection of the vaso-motor nerves, which have extended connections through the circulatory system.

It is worthy of remark here that the sensation of pins and needles, so common in the hands and feet, after prolonged pressure on the axillary and sciatic nerves, is due to arrest of blood-circulation and paralysis of the vaso-motor nerves.

If there be any truth in the supposition that pain is connected with disturbance of the capillary circulation, and not with peripheral irritation of the cerebro-spinal axis, we should expect to find it absent, or at least playing a very subordinate part in the causation of tetanus. It is a remarkable fact that pain and inflammation are generally absent from the wound which causes tetanus. Often, indeed, tetanus shows itself when the wound is kindly healing, or has healed. This fact has attracted the attention of most authors on this subject, and John Hunter, amongst the rest, remarks, that the irritability of the nervous system which developes into tetanus is not due to the presence of inflammation, since it appears only when the latter has been removed. It would appear from the records of tetanus that there is no connection between the frequency of tetanus cases and the painfulness of wounds. Neuralgias affecting, most painfully, the peripheral extremities of various nerves, never end in tetanus. The same thing may be said of gout. Neuromata, or subcutaneous tumours, are not known to cause tetanus; yet these are amongst the most painful affections to which man is subject.

I believe then, in answer to the question proposed at the head of this paper, that we may leave pain aside, at least for the present, and confine ourselves to the study of tactile irritation to elucidate the etiology of tetanus; not that I would exclude pain entirely, but because I think that it plays a subordinate part.

Before leaving the subject of neuralgia, it may be noticed that the late F. E. Ainstie, M.D.,[1] instances a number of cases of neuralgia resulting from injuries of various kinds to mixed nerves; even foreign bodies, as a bit of glass, have been entangled in the nerve, and caused neuralgia for months, but never tetanus. He makes the following comment: "So far as I know, it is only when a nerve-trunk of some size is injured that neuralgia is a probable result. Wounds of the small

[1] Reynolds' 'System of Medicine,' vol. ii. art. "Neuralgia."

nervous branches of the fingers, for instance, are very seldom followed by neuralgia. I have no statistics to guide me as to the effect of long-continued *irritation* applied to one of these small peripheral branches, but it is probable that that might be more capable of inducing neuralgia. As far as my own experience goes, however, it would appear that a more common result is convulsion of some kind, from reflex irritation of the cord."

It is observable that the nearer we get to the peripheral extremities of the nerves, the more markedly reflex is the effect of irritation. A cold plunge that sets a man shivering and shaking, and his teeth chattering—mark the tetanic symptoms—affects only the peripheral extremities of the cutaneous nerves.

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METHODS OF PREPARING, DEMONSTRATING, AND EXAMINING CEREBRAL STRUCTURE IN HEALTH AND DISEASE.

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II.

Physical Properties of Grey and White Matter.

In the introductory chapter to his great work on Pathological Anatomy, Rokitansky has said:—"Just as there is a general and a special anatomy, physiology, pathology, so there must in like manner be a general and a special pathological anatomy. The former treats of anomalies of organisation, the latter of the special anomalies of individual textures and organs." It appears to me that in the examination of the diseased brain this natural classification must be kept in view. The student should be taught to appreciate the various general anomalies of organisation as they present themselves in the brain; and the methods to be adopted in estimating the degree, extent, and significance of such physical deviations. It is not, of course, within our scheme to detail the various diseases of texture to which the brain is subject, but rather to place the student in possession of those indications of healthy and diseased tissues which are presented by alterations in the physical properties of grey and white matter. The elements of a physical diagnosis, so to speak, are placed at his disposal, and hints thrown out for his guidance, where it is thought a wrong construction might be attached to the physical signs.

It will be found, however, convenient to illustrate the more profound anomalies in consistence, colour, specific weight, and other physical qualities by reference to some of the special diseases of texture to which the nervous centres are liable, and

[1] 'Pathological Anatomy,' Sydenham Soc., vol. i.

which may serve as typical cases wherein the student may acquire a practical acquaintance with the methods of physical research.

The fundamental physical properties of the nervous tissues with which it is necessary we should acquaint ourselves are briefly as follows:—Consistence, colour, volume and weight, both absolute and specific. Each in turn will now claim our close attention.

A. *Consistence.*—The firmness, solidity, or consistence of a texture depends upon the cohesive force exerted by the individual elements of its mass, and to which are due the varying degrees of toughness, hardness, and resistance on the one hand, or of softening friability and liquefaction on the other hand, to which they are liable. The consistence of a compound tissue so complex as the brain is necessarily subject to very great variations, since its individual constituents vary amongst themselves greatly in their physical properties. An organ composed of nerve-tubuli, nerve-cells, of a connective framework and an elaborate system of arteries and veins, the whole pervaded by nutrient fluid, must be subjected to great modifications in consistence due to alterations in the relative proportions of one or other of the constituents. Hence it is that we get such variety in the structural firmness of brain-tissue, not only in different animals and in the brain of man, but in different regions of the same brain. It is this textural difference betwixt grey and white matter which explains their varying degrees of consistence, and the same explanation pertains to the firmness of the occipital as contrasted with the frontal lobe; of the central grey ganglia as contrasted with the grey envelope or cortex of the cerebrum. The grey cortex, consisting of a vast assemblage of nerve-cells and their protoplasmic extensions, imbedded in a most delicate web of connective tissue, with a complicated vascular apparatus, has a far lower degree of consistence than the white matter which owes its solidity to large medullated nerve-fibres and an abundant and coarser connective matrix. A moment's consideration will suffice to indicate to the student that the forces which in disease modify textural cohesion must come from *without* or from *within* the structural elements; in other words, the tissue

elements may be forced asunder by fluids pervading the texture and by the intrusion of adventitious bodies, or by nutritive changes in the elements themselves, whereby a quantitative or qualitative transformation may be induced. *What conditions, then, modify the normal consistence?*

1. The forcible infiltration of nervous texture by serous fluid, by plastic exudates, or by blood, will in the first place tend to destroy all textural cohesion, and consequently produce reductions in consistence; whilst further changes in the effused blastemata due to organisation will result in increased firmness or induration.

2. Chemical changes in the constitution of the individual elements, according to their nature, may tend to produce increase or reduction of consistence, e.g., fatty, amyloid, and calcareous degenerations, the nutritive anomalies due to inflammation, decomposition of the structural elements due to putrefactive changes.

3. Apart from the above qualitative anomalies of nutrition, we must not overlook the quantitative element which sometimes exists alone or in combination with the former. Such are the forms of hypertrophy and atrophy due to an increase in size and number of constituent elements on the one side, and on the other, in the decrease in size, number, and diminution in the number of the tissue elements.

The student must guard against the fallacy of regarding density and consistence of a tissue as in any way mutually convertible terms, for although in a large number of cases increased density may co-exist with increased consistence of the brain, conditions occur where with increase of density there is really a diminution in consistence. Thus, in inflammatory conditions of the brain-tissue where plastic exudates have forcibly reduced the cohesion of the mass, the specific weight of the part is notably increased. Hence the specific gravity of cerebral tissue will afford us no exact and reliable guide as to degrees of consistence; and, in fact, we have no more exact gauge of consistence of texture than the rough-and-ready methods afforded by the sense of sight and touch. How do we judge of the relative cohesion of texture?

a. By the eye we note deviations from the natural compactness of an organ, or the maintenance of its normal contour. Contrast the brain of a senile dement or of a general paralytic with the compact firm brain of an epileptic; or the same organ in warm weather and in a state of incipient decomposition with the recent brain removed from the skull in cold frosty weather.

b. By the sense of touch we note relative resistance to pressure and the compressible and lacerable character of the tissues. Especially note this with respect to the central commissural tracts, basal ganglia, fornix and septum lucidum.

c. By its resistance to a graduated force, such as the rough gauge afforded by a stream of water falling from a variable height (applicable only to cases of much reduced consistence). Any case of white or inflammatory softening may thus be tested.

d. By its resistance to section (applicable to the slighter degrees of reduction and all degrees of increased consistence). Contrast the resistance to section presented by the medulla, pons, cerebellum, cerebral hemispheres, and ganglia at base.

Observe that we gain by these means not only a satisfactory (although approximate) gauge of consistence, but that the methods vary in their relative values in different cases. The student should take every opportunity of rendering himself perfectly familiar with the general consistence of healthy brain prior to post-mortem change. Let him remove a normal brain for careful study, and note the following points:—

The Normal Brain.—Observe that upon removal from the cranial cavity it preserves its normal contour, although it has lost its natural support—the cranial bones. It is plump, rounded and compact, giving a general impression of firmness and solidity, ere we gauge its consistence further by handling. The hemispheres are closely approximated by means of the strong commissural band—the corpus callosum, showing no tendency to fall apart from rupture of the commissure, such as is so frequently seen in the degenerated and diseased brain.

Upon separating the cerebral hemispheres with the hand the corpus callosum appears intact, and offers fair resistance to the blade when dividing the hemispheres along this course. The individual lobes maintain their characteristic forms, their salient margins and relative positions; each convolution remains firm, plump, and in close contact with its neighbour, whilst the arachnoid investment gives a uniform, smooth, glossy aspect to the consolidated organ beneath. A section across the centrum ovale reveals a similar condition—observe the general solidity of the parts; how perfectly relative positions are maintained; the absence of any gaping of the sulci; observe also that the white substance cuts with a clean section, and shows no tendency to cling to the blade. Next open up the lateral ventricles, and observe the rounded firm aspect of the intraventricular portion of the corpus striatum and thalamus. Examine the fornix and septum lucidum—parts especially prone to softening in certain affections of the brain. Pay special attention to the relative consistence and size of the corpora quadrigemina, the cerebral peduncles, the pons and medulla. Now we must be prepared to meet with all varieties of consistence betwixt the above-described normal firmness of health and extreme alterations produced as the result of disease. The consistence, as we shall illustrate further on, may be so far reduced that the cerebral substance may be perfectly diffluent, and may be poured away like thin cream; or, on the other hand, it may possess the firmness, aspect and character of the hard-boiled white of egg; or even cause the knife to creak in cutting through patches of almost fibro-cartilaginous induration.

Reductions in Consistence.—Let us now consider briefly the appearance and significance of those abnormal conditions of cerebral tissue which lead to softening, dwelling only upon those which are most likely to present themselves frequently to the student's examination. The conditions best illustrative of reduced consistence are the following:—

1. Putrefactive changes.
2. Softening as the result of disease.
   a. White softening.
   b. Yellow or gelatinous softening.
   c. Red or inflammatory softening.

3. Simple œdematous conditions.

1. *Putrefactive Changes.*—Now these changes in the brain occur sooner or later after death, according to the temperature and humidity of the surrounding atmosphere, modified, of course, by the morbid conditions of the cerebral texture and contained fluid. It is on this account that the report of an examination of the brain should invariably be accompanied by a statement of the number of hours after death at which the examination was made, the average temperature of the post-mortem room, and the condition of the atmosphere as to humidity. Fremy, in an elaborate research in 1841 ('Ann. Chim.' 2, 463), arrived at the conclusion that the ordinary form of softening of the brain was analogous in its production to the putrefactive process occurring post-mortem; an opinion which has not been supported by later researches into the chemistry of the brain. The student should learn to discriminate between the softening due to this cause and that due to morbid alteration of texture. He will be guided here by observing that the softening is general throughout the encephalon, and attended by much blood-staining by the evolution of offensive gas, and the presence of the latter within the blood-vessels and beneath the membranes. He must also take into account the absence of any morbid condition tending to produce softening, and the conditions, atmospheric and otherwise, to which the brain has been exposed since death. It is essential also to keep in mind the fact that various diseases predispose to early putrefactive changes whilst the mode of death may expedite or defer the same.

How are these putrefactive changes to be avoided?

To retard putrefactive change when the brain cannot be immediately examined, it should be placed in a cold room with a damp cloth thrown over the hemisphere to prevent desiccation of the outer cortical layers; or better still, it may be enclosed within an ice safe. The student can readily extemporise for his own use such a safe, by placing the brain in a jar or box which fits into a larger box, leaving a space of an inch and a half around between the outer and inner box, into which ice is packed. The outer vessel must be thickly and completely enclosed in felt. Such a contrivance he will find

of great service in the summer months, when it is difficult to keep anatomical subjects fresh for many hours together. Portions of brain reserved for microscopic examination should be transferred immediately to methylated spirits or Müller's fluid, or better still, to methylated spirits coloured of a dark sherry tint by tincture of iodine. For the fresh methods of examination should any delay occur, we must trust to the ice safe for the preservation of our material—preservative fluids being scarcely admissible. As regards the latter, however, the strong solution of acetate of ammonium recommended by Sankey[1] is perhaps the best which can be used (sp. gr. 1·040).

Indications of Softening.—A general reduction in the consistence of the brain will usually reveal itself at first sight to a mere superficial glance. The whole brain assumes a flattened squat appearance, and the hemispheres diverge. The student will find the former due to extension of softening to the central portions of the hemispheres which support in the normal state the convolutionary surface folded over them, whilst he observes the commissural band connecting the hemispheres (corpus callosum) tends to split asunder in a longitudinal direction, i.e. across the course of the great bulk of its softened fibres. Proceeding to handle the brain he finds the convolutions flabby to the touch and presenting less resistance to pressure—the whole brain is less able to resist the ordinary force of gravity, and is therefore strongly contrasted with the plump, erect, and compact aspect of the healthy brain. Let the student examine the brain of an advanced general paralytic and he will find this condition well represented. Cut across the hemisphere of such a brain so as to expose the *centrum ovale*, and it will be observed that the brain-tissue clings to the blade with unusual tenacity unless the latter be kept constantly wet by water or spirit. As a result the cerebral tissues both grey and white are lacerated and tear away in shreds, leaving an unmistakable softened rottened aspect of the surface of the section. This tearing away of brain-tissue is apt to give one a false idea of the *comparative*

[1] 'New Process for Examining Brain Structure,' West Riding Asylum Reports, vol. v. p. 192.

coarseness of texture; thus in the brain of a general paralytic, the adherent, thickened membrane and softened cortex cannot be cut without leaving a coarse irregular surface, unless the precaution be taken of using a very sharp blade and keeping its surface constantly wet. Let us now examine more carefully the special form of softening, known as white softening.

2. *White Softening of the Brain.*—We may meet with this condition as one generally diffused through a hemisphere or in patches limited to a convolution, the area of softening being often no larger than a pea, although more extensive tracts are usually involved. In the first place, suppose we have before us a brain in which the greater part has thus suffered. The affected hemisphere presents to the touch a soft, boggy feel, sometimes communicating an almost tremulous, fluctuating sensation to the finger if the disintegration be extreme. The brain will be with difficulty removed, the disintegrated tissue tending to burst through the softened walls which confine it, and more especially is this the case in the neighbourhood of the anterior perforated space and commencement of the Sylvian fissure. All unnecessary manipulation of such a brain should be avoided, as it is attended with danger to the textural continuity of internal parts, which should also be examined at as early a stage as possible. Observe the lateral divergence of the hemispheres and their flattened aspect above. By gently drawing the hemispheres apart and introducing the blade of a knife into the longitudinal fissure a section may be made outwards across the affected hemisphere, and thus removing the vault we find the centrum semi-ovale occupied by a mass of broken-down medulla—all normal cohesion being destroyed by the presence of a large quantity of interstitial serous effusion. The disintegrated substance will probably have the appearance of soft watery or creamy pap readily raised upon the blade, or it may be absolutely diffluent, flowing away as soon as the resistance offered by the confining grey cortex is overcome. It may, however, retain a certain amount of cohesion, yet break readily and wash away on placing it beneath a stream of water. The ganglia at the base may be implicated, and the septum pellucidum, grey commissure, fornix and corpus

callosum will generally be found softened or even broken down, the ventricle containing more or less serous fluid. Now such a brain as we have just been considering will be met with in acute hydrocephalus, and in thrombosis or embolic plugging of the main cerebral arteries. With such a case before him, the student should pay attention to the following points:—

The degree of softening.—Does it resist a stream of water?
Does it render a stream of water milky or break down readily under it?
Is it from the first diffluent?

Extent of area involved.

Œdematous condition of the parts involved.

Turbidity or clearness and colour of effused fluid in the ventricles.

In noting the degree of softening, learn to appreciate how very short a period is requisite after arrest of blood supply, for the production of extensive nutritive changes. With what object do we endeavour accurately to map out the *extent* of the destroying lesion? Our object is to detect that portion of the vascular apparatus involved if the softening be due, as it most frequently is, to plugging of a large branch. This the more frequent source of extensive white softening requires therefore for its full comprehension an acquaintance with the vascular areas of the brain.

On noting the appearance of the serous effusion in the ventricles, so often accompanying white softening, the student will be guided towards concluding as to the presence or not of inflammatory action in the meninges or in the substance of the brain itself. Thus if the fluid be more or less turbid and discoloured (as the result of a plastic exudate and macerated cerebral substance), we may reasonably suspect an inflammatory condition, and expect to find upon microscopic examination exudation corpuscles, compound granule masses and other products of inflammation. Such are the conditions found in acute hydrocephalus, distinguishing it from a purely non-inflammatory form of hydrocephalus, which occurs where the fluid, though perhaps slightly turbid from broken-down cerebral tissue and shreds from the macerated lining membrane

of the ventricles, presents no inflammatory material amongst the débris.

Limited Foci of Softening.—Acquaintance with the peculiar arrangement of the cerebral blood-vessels soon leads the student to infer that very minute tracts of softening may result as the effect of thrombosis or embolism, the area involved being dependent upon the size of the clot and the site of its arrest or formation. It is necessary that the mechanism of white softening of cerebral tissue, as a result of thrombosis or embolism, be thoroughly understood. The process may be best elucidated by the diagram below.

DIAGRAM ILLUSTRATIVE OF THE EFFECTS OF EMBOLIC PLUGGING (AFTER RINDFLEISCH).

a a. Portion deprived of its blood supply by the Embolus E. A. Artery. V. Vein filled with blood-clot. The arrows indicate the collateral channels which lead to a hyperæmic zone around the occluded vessels.

Beyond the obstructed artery is the wedge-shaped area of its distribution, now anæmic and consequently deprived of its functional power. Below the embolus are seen swollen branches, which tend to establish a collateral circulation. If this fails, we get as a result engorgement of the latter vessels, and a congestive vascular zone surrounding the central mass. The tissue here becomes swollen and œdematous and minute hæmorrhages are apt to occur, whilst the whole central and peripheral texture becomes broken up by the effusion, and a true necrosis occurs of the tissue forming the area of distribution of the nutrient branch which has been plugged.

All inflammatory foci have as a result the production of a similar congestion and œdema of the surrounding texture,

accompanied by white softening; and as new growths or tumours are frequently the site of such inflammatory states, we may find them imbedded in a cavity containing broken-down tissue.

In addition, therefore, to the appearances which the student has been directed to look for in white softening of the brain, let him also note—

a. Any obstruction to the vascular supply by thrombi, emboli, or adventitious products pressing upon the vessels from without.[1]

b. The establishment of a collateral circulation.

c. The presence of inflammatory foci, to which the white softening is secondary.

Yellow Softening.—In the course of his pathological studies the student will frequently meet with appearances which, whilst significant of cerebral softening, differ much from that just described. He will find frequently in the white substance of the hemispheres, less frequently in the cerebellum, and rarely on the convolutionary surface of the cerebrum, a focus of softened tissue, varying in size, but scarcely ever larger than a hen's egg, and characterised by its bright straw-yellow colour and soft gelatinous consistence. Pressure causes a yellowish fluid to exude, and when cut across it is found to be somewhat sharply defined from the pale swollen and œdematous tissue around. The student will recognise in these softened areas the lesion termed yellow softening of the brain.

Pushing on his examination, he will discover that these spots may be primary, or, on the other hand, secondarily induced around adventitious products, such as tubercle, cancer, cysts, hæmorrhages, or around a patch of inflamed tissue. If secondary to adventitious growths, there is, as Rokitansky points out, an intermediate zone of red softening betwixt the growth and the yellow softened exterior.

It is important that these patches should not mislead the student. He must from the outset regard them as identical in nature with the first form of softening described, the difference in colour being due, according to some, to altered blood-pigment, but according to Rokitansky, to a peculiar chemico-pathological process.

[1] Plastic exudates, tubercle, syphilitic gummata.

The following facts are to be noted:—

a. The fluid is usually intensely acid.

b. There is little or no vascularity surrounding the patch.

c. Every degree of tint may be found, from white softening up to the bright yellow of typical yellow softening.

d. Colour differs from the rusty or ochre-yellow tint seen in old apoplectic cavities, &c.

e. Microscopic examination fails to reveal inflammatory products.

Red or Inflammatory Softening.—My reader will now be prepared to infer that profound alterations in consistence may attend inflammation of the nervous structures, and he will find little difficulty in recognising foci of inflammatory softening. He will occasionally meet with portions of acutely inflamed tissue in the medullary strands of the cerebrum, varying in size from a hazel-nut to that of an orange, or still larger; yet by far the more frequent site of such inflammatory foci will be the grey cortex of the cerebrum and the basal ganglia. Examine a brain which presents a focus of inflammatory softening in the medulla of one of its hemispheres. Note the following facts:—The *softening of texture* is accompanied by modifications of colour and general moisture. Thus the increase in vascularity gives the affected part a streaky red aspect, often profusely besprinkled by puncta vasculosa; the œdematous infiltration of texture is recognised in its swollen and moist aspect. Where the engorgement has reached a high degree, note the numerous minute extravasations of blood, the streaked or punctuated capillary hæmorrhages.

Now the student must be fully impressed with the fact that all the above characters may be developed in the course of extreme congestion. What impresses upon the affected part the stamp of inflammatory action is the presence of exudates, which modify the above described appearances in two directions:—

a. The dark red becomes of a paler and more uniform hue, whilst exudations of lymph still further modify the aspect.

b. Alterations in consistence occur; rapid softening or solutions of textural continuity varying with the plasticity of the exudates.

Observe, therefore, that the essential features of red inflammatory softening are profound alterations of consistence, associated with the pouring out of inflammatory exudations.

In advanced stages of red inflammatory softening, the student will, therefore, find the tissues broken down into a reddish pulp, or having a brownish or rusty aspect, washing away freely when held beneath a stream of water. He will also note the results of swelling from congestion and œdema, viz., the flattening of the convolutions of the hemispheres from pressure against the internal table of the skull, and a well-marked prominence of the surface of a section of the inflamed and œdematous textures.

Examine a patch of inflammatory softening in the cortex, and note :—

Inflammatory foci situated in the cortex will implicate by contiguity the superimposed membranes. Observe in these cases that the grey matter is more deeply coloured, and is still more swollen, moist, and softened than when the white matter is involved. The far greater vascularity and the naturally looser texture of the grey matter account for this difference. Next direct attention to the state of the membranes. The pia mater cannot be removed without peeling off with it a layer of the softened cortex, whilst the meshes of the membrane are found infiltrated with inflammatory products and are both thickened and œdematous. As regards these apparent adhesions of pia mater, observe that the brain substance may tear away with the removal of the pia mater from two causes :—

1st. Mere unnatural softening and loss of textural cohesion of the grey matter, without any true adhesion to the membranes as in early inflammatory and congestive stages, &c.

2nd. Actual adhesion, inflammatory in origin, may be the cause of this tearing of the cortex.

How are we to distinguish the lacerable cortex in the former case from the genuine inflammatory adhesions of the membranes? In the former case the surface left by the irregular shreds torn off is soft, pappy, and uniformly smooth or homogeneous; in the latter case the surface is studded with numerous perforations—the apertures of perivascular

channels from which the blood-vessels have been withdrawn. These channels will be seen much dilated from the prior engorgement of the blood-vessels, and the surface generally has a peculiar worm-eaten appearance.

Fallacy to be avoided.—The student must not regard all cases of encephalitis as necessarily attended by injection and discoloration of tissue. The larger number of cases, in fact, show little or no discoloration; and even where softening has advanced to an extreme degree, the naked eye fails to appreciate in the uniformly dull, white pulpy material its inflammatory origin.

What criterion have we here for determining the nature of the lesion? Recourse must be had to the microscope and specific gravity bulbs.

The *microscope* shows us nuclei, nucleoli, pigment, and compound granule cells amongst broken-down cerebral tissue infiltrated with leucocytes and effused lymph along the course of the blood-vessels. The specific gravity test (to be hereafter described) will indicate an invariable increase in specific weight where inflammatory exudation has occurred. On the other hand, non-inflammatory white softening shows an invariable reduction in specific gravity.

By the freezing methods it will be possible to obtain fair sections through inflammatory patches of greatly reduced consistence, and much valuable information will be thus obtained; but in all cases of extreme softening all that can be hoped for is obtained by raising a little of the creamy pulp upon the scalpel, and transferring it to a slide and using a thin glass cover. Sections through the membranes and cortex, in cases where these are involved, will prove highly instructive, and should never be neglected. They should be studied in the fresh state, and the student become thoroughly familiarised with the morbid appearances ere he attempts to mount stained sections of such lesions for permanent preservation. Coarse blood-vessels, coursing through the inflamed patch should be drawn out and examined microscopically, noting abnormal conditions of their coats and sheath.

(To be continued.)

Critical Digests and Notices of Books.

Maladies de la Moëlle. Par A. Vulpian, Doyen de la Faculté de Médecine, Membre de l'Institut, etc. etc. Grand 8°. Paris: Octave Doin; pp. 512.

The volume before us contains a series of lectures on some of the diseases of the spinal cord, delivered by the author in his capacity of professor of experimental pathology. M. Vulpian's great authority, both as a physiological and clinical observer, as well as his felicity of expression, make the reading of this book a duty as well as a pleasure to all those interested in spinal pathology. We regret, however, that he should have preserved the form of lectures in the present work. The subject-matter is wofully cut up; and yet considerable changes must have been made in the original 'Leçons.' For instance, Lecture XIII. occupies twenty pages, devoted to the incipient stage of locomotor ataxy; whereas in Lecture XIV. the first stage is concluded, and the second and third discussed and illustrated with cases, the whole occupying two hundred pages!

Experimental pathology, though bound within very narrow limits, and unable to reproduce in animals most of the diseases observed in man, has thrown floods of light upon certain obscure symptoms of spinal lesions. But it has done more than that: it has given origin to a stricter spirit of clinical observation and educated the mind in the framing of scientific hypotheses, and so rendered possible the solution of problems to which its methods were not directly applicable. Indeed it is doubtful whether the cultivation of this mental discipline is not of greater value than the actual establishment of facts; and it is to be regretted that the defence of vivisection against the

blind fury of the sentimental fanatics who revile it and its devotees, should not have been more generally based upon this higher ground, rather than its legitimacy vindicated by an appeal to the individual discoveries to which it has led. Though the leading thought of Prof. Vulpian's work is the explanation of the various symptoms observed in the diseased human cord through the data of experimental inquiry, he has given a very large share to the purely clinical aspect of the question; and though the various diseases touched upon in the present volume are not all as fully discussed and illustrated with typical cases as locomotor ataxy, for instance (which occupies about half the book), and the work has no claim to be a systematic exposition of spinal complaints—yet theory is so constantly verified by references to practice, that no one will arise from the perusal of these pages without being a better physician as well as a wiser pathologist.

The first subject discussed is compression of the spinal cord, sudden or gradual. The symptoms in both cases involve, eventually at least, similar consequences; it may be observed, however, that in acute compression sensory troubles are usually not conspicuous. Chronic compression occurs as a consequence of a variety of pathological states, such as growth of various kinds, thickening of the dura mater, effusions into the canal, etc. Caries of the vertebræ, or Pott's disease, offers a good example of it. The actual cause of the compression here has often been stated to be the deformation of the column; but this is certainly erroneous in the great majority of cases, for on the one hand, compression is sometimes present where no deformation is noticeable; on the other, considerable deformation may occur without any signs of compression being developed. The compression is due to an external pachymeningitis, as shown by Michaud. The external layers of the dura mater are inflamed by contiguity, and become the seat of a fibro-nucleated proliferation, and a caseous layer usually results from the retrogressive changes in the neoplasia. This appears to the naked eye as if merely superimposed upon the internal layer of the dura mater, but the microscope easily displays its true nature. Prof. Vulpian does not agree with those who refer all the pain often associated with this pachy-

meningitis to pressure upon the posterior roots. He appeals to the results of his experiments, which have shown the dura mater itself to be acutely sensitive, especially when inflamed. Of course it would be unsafe to base this opinion upon mere manifestations of suffering on the part of the animal experimented upon. But we have two ways of proving the irritation of sensory nerves; the one, by the increased vascular tension with heart-beats diminished in number, but increased in strength; the other, by the dilatation of the pupil. By both these methods we can prove the dura mater to be sensitive.

The acuteness of pain in Pott's disease may induce a pseudo-paraplegia, which, however, with advancing pressure, is soon converted into a true paraplegia. This interference with the transmission of voluntary impulses through the cord is often entirely dissociated from any corresponding anæsthesia—an important diagnostic feature in cases of compression. To explain this fact by the assumption that pressure is mainly localised on the anterior aspect of the cord is erroneous. It is much rather due to the fact that the conductivity of the grey matter, alone necessary for the preservation of sensation, is less readily destroyed than that of the white fibre columns. There are, however, many cases where hypæsthesiæ and paræsthesiæ of varied character testify to some alteration of the grey matter.

The increase in the reflex activities of the lower segment of the cord, when cut off from the higher centres, has been attributed by Setschenow to the suspension of the inhibiting influence of a special centre, which he placed in the isthmus. But, as Vulpian shows, direct stimulation of the isthmus does not suspend the spinal reflexes; and their exaggeration increases with the distance from the brain at the point at which the cord is cut. With reference to the augmentation of reflexes, amounting to tonic rigidity, observed in the later stages of compressive paraplegia, we note that it is frequently preceded by a period of muscular resolution. Possibly, descending sclerosis plays a part in its causation; but we may hope that a cure is not impossible even when rigidity is well marked.

According to the locality and nature of the lesion (irritative or paralysing) the vasomotor and trophic symptoms (as well as

those connected with micturition and defecation) will vary. Of special interest is the compression of the cervical enlargement, which brings about brachial paraplegia. In a frog this can be easily illustrated by inserting a small piece of wood along the spinal canal. The animal then swims easily with its legs, while the arms hang helplessly down. Generally speaking, every compression acts most markedly upon the nerves originating at the point compressed, the explanation of this fact being possibly that its influence is more readily exerted upon those motor fibres emerging from the grey matter to form the anterior roots, than upon those constituting the lateral bundles from the cerebral centres.

A paradoxical result often observed is that compression of the mid-dorsal cord frequently involves paresis of the arms (recurrent paralysis). In connection with this point it must be remembered that the vasomotors of the upper extremity arise from as low as the level of the seventh dorsal pair. There is always some degree of myelitis in Pott's disease, and this may give rise to more or less secondary damage in the cord, ascending or descending.

Compression of one-half of the cord gives rise to the well-known symptoms described by Brown-Séquard. It must be remarked that in spinal, as well as cerebral hemiplegia, the arm is always more involved than the legs. Professor Vulpian scatters to the winds the fanciful hypothesis so popular in some quarters, and which attributes so much and varied influence to disturbed circulation in the cord. Experimentally and theoretically, he demolishes that "deus ex machinâ" of the pathologist in trouble—"vasomotor changes"—and proves his assumptions to be mere "post hocs." Congestion of the cord may, however, produce symptoms in certain morbid conditions; and he describes a case of suspected varices of the organ where ergotine was of great service. With reference to anæmia, it is impossible to conceive the occurrence in the human subject (except perhaps through a sudden diminution of atmospheric pressure) of a paralysis similar to that produced in animals by artificial embolisms of the spinal vessels; and unless with Hammond we believe in the possibility of anæmia of individual bundles of fibres, how are we to explain the

persistence of sensation in those cases where paraplegia has been attributed to vasomotor spasm?

We rapidly pass over several topics which the author discusses ably, but without adding much to our previous information, noticing only on the way the importance he attributes to the rich nervous supply of the pia mater and arachnoid, especially on the posterior aspect of these membranes. These facts may explain not only the pains of arachno-lepto-meningitis, but also perhaps the greater intensity of the inflammatory process generally prevalent in the posterior surface of the cord. Patches of chronic arachnitis often found post mortem may, he thinks, explain certain obscure spinal symptoms; but, notwithstanding his efforts, he has not been able to trace very clear conclusions as to their clinical import.

Professor Vulpian has been the first to establish a distinction between the diffuse and the systematic forms of myelitis, and to explain their pathogeny by assuming (a view now very generally adopted by French writers) the former to consist in a primarily interstitial, the latter in a primarily parenchymatous inflammation. Among the usual causes of acute diffuse myelitis are propagation (from meningitis), traumatism, tumours, fatigue. He fully discusses the ætiological bearings of cold. It is clear that since meningitis is as frequently produced by exposure to cold as myelitis, we cannot assume in the latter a direct influence from the periphery through the afferent nerves. There must be a reflex action transmitted through the sensory (trophic) nerves supplying the meninges and the connective tissue of the cord itself. The impression made on the surface disturbs the nutritive functions of the centres with which the part is in relation, and this is manifested by an inflammatory process in the tissues supplied by the same centres.

What individual tissue or organ will become the seat of the morbid change depends, of course, upon some idiosyncrasy or special predisposition. The theory is applicable not only to myelitis, but to pleurisy, peritonitis, nephritis, &c.—in a word, to all inflammations due to the effects of a chill to the surface. Experiments show how readily myelitis can be induced by irritation of peripheral nerves; and here we have a satisfactory explanation of the so-called reflex paralysis and

atrophies, according to the author. The administration of lead and bromide of potassium to dogs has also been followed with marked meningitis. We pass over the discussion of acute diseases in their relation to myelitis, as we have had lately the opportunity of reviewing Dr. Laudouzy's exhaustive treatise ('BRAIN,' X.). We need not say anything of the pathological anatomy and of the sensory and motor symptoms of myelitis, as the author does not contribute any new facts with reference to them. The vasomotor centres may be excited or paralysed. In the first case, should the constriction of the vessels extend to the venules, the limbs become cold and pallid; if confined to the arterioles they are cold and livid—precisely what we find obtaining in Raynaud's asphyxia of the extremities. Paralysis of the vasomotors is characterised by redness and heat at first, followed by blueness and coldness due to the increased cutaneous radiation and the atrophy of the surrounding tissues. Among the trophic disturbances the author discusses the "acute decubitus" (a misnomer he thinks), which he attributes to the united effects of the several causes at work, and not to a specific action of the disturbed innervation. The cystitis, which is often present, is explainable on similar principles. The innervation of the lungs, stomach, intestines, &c., if disturbed, may set up various morbid processes in these organs.

The intensity, extent, and localisation of the myelitic process vary greatly; and to these variations correspond all the grades of the disease, from the apoplectiform type, which kills outright, to the lightest type, which betrays itself best by the slight paresis of a limb. The process, again, may attack the whole cord "d'emblée," or come under the designation of ascending or descending. Professor Vulpian incidentally draws a parallel between Landry's disease and acute ascending myelitis, and thinks that whilst there is as yet no pathological reason for identifying them, neither is there any ground for explaining the former by a toxæmic process: the question is still open. It must not be forgotten that several cases, recorded under the name of Landry's disease, are really nothing but more or less aberrant forms of myelitis, as the description of the symptoms clearly shows.

Among the various forms of chronic diffuse myelitis, we must first distinguish those in which the white matter is affected from those in which the grey matter is the seat of the morbid process; in other words, leuco-myelitis from polio-myelitis. Leuco-myelitis, again, may be cortical or annular, as in many cases secondary to meningitis, where a ring of white matter only is affected (mainly on the posterior aspect, where we have seen the meningitic process to be most intense), or spreads through the whole depth of the white columns, as is typically the case in general progressive paralysis. The pathological change in the connective tissue is the same in diffuse and in systematic myelites; but in the former the destruction of nerve fibres is very rarely as complete as in the latter, where, as above mentioned, a parenchymatous inflammation is assumed to be the primitive lesion by the French school of neurology.

In chronic diffuse polio-myelitis the alterations may be confined to the peri-ependymary region, a form which has been carefully described by Hallopeau as "peri-ependymary myelitis," and is characterised by the development of vacuoles, often filled with liquid. Michaud has stated that the lesion of tetanus is allied to that obtaining in this form of myelitis; and Professor Vulpian, in his experiments on the toxic effects of large doses of bromide of potassium, has found the paralysis of his dogs apparently arising from a similar peri-ependymitis.

True diffuse myelitis affects both the grey and the white matter, though not evenly, perhaps, nor universally. The symptoms of it will, of course, vary with the mode and intensity of its progression; and its diagnosis will be based upon the combination of the symptoms present, motor, sensory, trophic, reflex, &c., which will also frequently reveal the localisation of the pathological alteration in the cord. Duchenne has committed a serious error in separating under the name of "general subacute spinal paralysis" certain cases of diffuse myelitis, and giving them a rank co-equal to the anterior polio-myelitis he has defined with such masterly precision.

The second half of Professor Vulpian's volume deals with progressive motor ataxy, as the chief variety of systematic myelitis has unfortunately been named. Since 1862 our author has pleaded for the syphilitic etiology of tabes;

out of 20 patients 15 at least have had syphilis, according to him. Fournier (1876) gives a still higher percentage, 80 per cent. Gowers ('Diseases of the Spinal Cord,' p. 62), in a curiously-expressed passage, in which he does not mention these and other earlier observations, has espoused the same view. In a paper published in 1879 he "expressed the opinion that one-half of the cases there is a history of antecedent syphilis. The same opinion has since been expressed by Erb," &c., and he states that out of 16 patients lately examined by him 11 had had chancres, and many symptoms of constitutional syphilis. Against the actual causal nexus between syphilis and tabes many arguments can be urged. First, the discrepancy in the statistics of a large number of competent pathologists, who have not found a high proportion of specific antecedents to tabes (Westphal, for instance, who gives a much lower figure; and, besides, on the ground of numerous post-mortem examinations, doubts the syphilitic nature of the disease). Leyden rejects the theory altogether. Chauvet plainly says (1880), "Syphilis never will cause a primitive sclerosis of the posterior radicular zones." Again, it has been stated by Westphal, Bernhardt, and others, that syphilitic antecedents have never been traced by them in any female case of tabes. And yet would not the prevalence of the infection among prostitutes make them peculiarly liable to that manifestation of the disease? Finally, whatever may be said to the contrary, the utter failure of mercury and iodide in the treatment of tabes[1] is a presumption against the syphilitic nature of it. Do we not, in fact, look upon the successful administration of these drugs to patients suffering from symptoms of doubtful origin as an almost diagnostic sign? It would be strange indeed that tabes should make such a startling exception to the rule. All we are disposed to concede is that the syphilitic cachexia may increase the natural predisposition of certain individuals

[1] In a report of a paper read by Dr. Dowse ('Brit. Med. Journ.' Dec. 11) he is made to say that "every case of locomotor ataxis, with very few exceptions, may be traced to syphilis as a cause; and that every case of the disease was curable" if treated sufficiently early and vigorously. Happy doctor, happy patients! But we are at a loss to understand the rationale of this anti-syphilitic treatment: Bromide, ergot, counter-irritation, besides merc. bichlor. gr. ¼ per diem. Nerve-stretching appears to be the most promising form of treatment. Its success would be a fatal argument against the specific nature of the disease.

to tabes as it does to other intercurrent affections. We should remember, also, how the once universal idea of the relationship between tabes and sexual excesses has been shown to be fallacious. Another point to be noted is the author's opinion that there is a relation of cause and effect between hysteria and tabes in women. Would not rather both be best considered as manifestations of a neurotic tendency? A more important connexion is that of tabes with progressive general paralysis. Professor Vulpian gives a very full symptomatology, with copious illustrations, by means of well-selected and described cases, of the three periods of tabes. He lays special stress upon the invariable presence of sensory troubles; some anæsthesia is the rule; but the perception of cold is often singularly vivid and calls forth reflex movements. A full discussion of this part of the work, however, as well as that which treats of the pathological alterations, would lead us beyond our limits. Moreover, the author does little more than give an exposition of the already known facts. The last two lectures, however, deal with the pathogeny of the morbid phenomena, and deserve to arrest our attention for a while. The author is strongly of opinion that the nature of the primitive lesions is not a pure atrophy, but depends upon a morbid irritation or an inflammation of the axes-cylinders of the posterior fibres. The atrophy so widely prevalent among the affected parts is secondary. Some authors have propounded a doctrine opposed to this view. According to them the sympathetic was primarily affected; and upon this lesion followed some disturbances of the nutritive vessels of the cord, to which again the atrophy of the nervous elements was secondary. Unfortunately for this theory its first link is weak: the sympathetic has often been found absolutely healthy in advanced cases of posterior leuco-myelitis. But could not the disease be secondary to vascular changes setting up an intestinal myelitis? This is a more insidious argument; but it must be remembered that the posterior roots are atrophic also. How could this be accounted for upon this hypothesis?

The author develops his views with reference to the primitive morbid irritation, probably of inflammatory nature, of the axes-cylinders, with great skill and force. The secondary

lesions of the connective tissue are accounted for in a most ingenious manner. The axes-cylinders lose their vitality, and act to all intents and purposes as foreign bodies, or sequestra, and stimulate the surrounding tissues to an inflammatory reaction. Passing to the order of succession in which the lesions spread, and having explained how the inflammatory process set up in the adneural structures may be propagated to the meninges, Prof. Vulpian shows that the accepted theory of Charcot and Pierret does not remove all the difficulties. For how does the morbid process pass from the fibres of the external radicular zones, first to the rest of the posterior fasciculi, and second, to the posterior roots? If there be any fact well established in physiology, it is Waller's discovery of the trophic dependence of the posterior spinal fibres upon the posterior root-ganglia. Though unable to reconcile the conflicting data, the author visibly inclines to place in the posterior roots the starting-point of the morbid process. In an ascending direction sclerosed tracts may be followed throughout Goll's columns, which have undergone secondary change.

Whatever difficulties we meet in our attempts at an explanation of the various symptoms and their anatomical substratum, they vanish in presence of the impossibility of accounting for the pathogeny of that substratum itself. Clinical and experimental investigation here completely fail us.

It is commonly thought that the anæsthesia usually prevalent in tabes depends upon the sclerosis of the posterior columns. Schiff's experiments are relied upon as a physiological basis. But these are far from being above criticism, and Prof. Vulpian has failed to obtain the same results as the Genevese observer. Not only was no anæsthesia noted after section of these columns, but there supervened hyperæsthesia (as well as hyperalgesia). Hence it is only when the posterior roots are involved, that sensation can be diminished in posterior leuco-myelitis.

How is it that the tactile sensibility of a whole limb may be lost, whilst a number of sound fibres exist in the posterior roots? Are we then to conclude, to a special set of fibres for each quality of sensation—of touch, of temperature, of pain, &c.?

No, but simply accept the hypothesis (borne out by the fact, that more fibres enter the posterior ganglia from the periphery than leave it to enter the cord), that a sensation carried to the ganglion by one fibre is there spread over (so to speak) the whole root. Any deficiency in the number of fibres here will, therefore, diminish the intensity of the sensation. With reference to analgesia, it is only anæsthesia carried one degree further; pain, too, is but sensation carried one degree further. Painful anæsthesia is simply due to the psychical localisation in an anæsthetic limb of pains due to an irritative process of its central sensory tract.

The author incidentally discusses the anodyne effect of morphia injections along the course of the nerve to which the painful sensations are referred, such as the characteristic lightning pains. He attributes it less to the general absorption of the drug than to the centripetal effects of the local anæsthesia induced by the injection. In the same way local applications of mustard, chloroform, &c., allay the shooting-pains, not by reflex central vasomotor changes, as is so often presumed, but by some direct excitation of the ganglia.

In trying to explain these facts, as well as the various sensory disturbances common in tabes, we must remember two facts, that the fibres from the periphery to the ganglia are usually healthy, and that they have probably no direct continuation through the ganglia to the cord, but are interrupted there, the further conduction of impressions requiring possibly the integrity of the whole root. When, therefore, the number of fibres constituting that root is diminished, powerful and protracted stimulations may yet be felt when all weaker and shorter ones fail to be perceived. Central and peripheral anastomoses may also be brought in to explain the reference of sensations to wrong spots on the periphery. The author tries to explain the persistence, or even increase, of thermæsthesia by the special mode of central stimulation, which we must assume to be the condition of the perception of heat and cold when the existence of special fibres is denied. We know how the sensory tracts of the cord are sometimes stimulated by the passage of a hot or cold sponge along the vertebræ: hot and cold impressions, therefore, seem to have a peculiar

power of stimulating the spinal sensory elements under certain morbid conditions. With reference to the retardation of transmission of sensations (a phenomenon, by the way, the frequency of which we believe to be greatly exaggerated by some writers), it is not due to the diseased condition of the posterior cords, for it is not observed in animals on which these tracts have been destroyed, but rather to the diseased condition of the posterior ganglia, where, as above mentioned, the different fibres are probably interrupted. The posterior grey spinal matter, which is assumed to be æsthesodic, does not generally present sufficient signs of alteration to account for the delay.

It is rare that absolute anæsthesia prevails, even in advanced cases. Protracted and energetic faradisation often succeeds in restoring a limited and temporary sensibility at the spot where it is practised.

The last few pages of the book are devoted to the subject of the motor ataxia. It is unfortunate that more space was not devoted to this interesting and important subject. As everybody knows, two leading hypotheses occupy the field. The first assumes the disordered balance of muscular action to depend upon the want of co-ordinated spinal stimulus, due to the lesion of the intercellular fibres assumed to exist in the posterior columns; the second explains it by the impaired impressions from the periphery, and the deficient knowledge of the whereabouts of the limb, so to speak. In the first instance, the impulses are correctly given, but incorrectly obeyed; in the second they are incorrectly given, owing to mistaken information. To discuss these points would carry us far beyond our present limits. Prof. Vulpian accepts the second theory (known in Germany as that of Leyden), though open to very grave objections. His reason for rejecting the first is simply that the existence of the intercellular fibres has never been demonstrated anatomically. Here, as all through the book, we are made to feel the utter neglect with which too many French pathologists and physiologists treat foreign, and more especially German, science. No doubt the modern school of French neurology is shedding splendid rays of light in every direction. But there are luminaries of the first

order elsewhere, and completeness is a quality as well as originality. Could not M. Vulpian combine the former with the latter to a greater degree? He often quotes Duchenne and Pierret; why not add the names of Erb and Flechsig? Has not electro-diagnosis been raised by the former to a far more dignified position than that where Duchenne had placed it? and have not Flechsig's researches on the development of the cord opened a new era in its physiology and pathology? Yet in spite of these deficiencies Prof. Vulpian's last work remains fully worthy of its author's great reputation.

A. DE WATTEVILLE.

The Science and Practice of Medicine. By WILLIAM AITKEN, M.D. Edin., F.R.S. Seventh Edition. London: Charles Griffin & Co., 1880.

A BOOK on the Science and Practice of Medicine which extends to 2381 pages of unusual amplitude, and has gone through seven editions in twenty-three years, is beyond the reach of criticism in a double sense. It baffles it, by its comprehensive immensity, and it is independent of it by its assured reputation. We feel that it would be impertinent to do more than welcome Dr. Aitken's great and standard work in its new, enlarged, and remodelled form. Excellent from the beginning and improved in each successive issue, this work has now, with vast and judicious labour, been brought abreast of every recent advance in scientific medicine and the healing art, and affords to the student and practitioner a store of knowledge and guidance of altogether inestimable value.

It is in those sections of Dr. Aitken's book that are cognate to the scheme of this journal, that the most important alterations and extensive additions have been made in the new edition. Undismayed by the magnitude of the task, the author has vigorously grappled with the immense and confused crowd of facts bearing on diseases of the brain and nervous system, that have been brought to light of late years, and has succeeded marvellously in reducing them to order, and in assigning to them their just rank and station in the

body of previously ascertained knowledge. So comprehensive and accurate is the survey taken of this department of medicine, that the first 530 pages of the second volume in which it is comprised would, if printed separately, form perhaps the best text-book in our language for the student of neurology and insanity. A masterly and philosophical review, characterised by the precision of the specialist and the breadth of the catholic physician, is presented in those pages of the varied phenomena connected with morbid conditions of the nervous system in their relations with anatomical structure, chemical composition, physiological uses and pathological changes. An introductory sketch of the direction taken by modern inquiry into the functions and diseases of the nervous system, is followed by a general outline of the crude anatomical constituents of the nervous system, and by a detailed account of the histology and arrangement of the membranes of the brain and cord, and of the specific gravity of the brain, and its weight at different ages and in different races and conditions. Next comes a description of the external configuration of the brain, of its several convolutions and minute structure, and of the composition and structure of the other encephalic centres, as complete and minute as is to be found in any handbook of anatomy. The succeeding sections deal with the vascular supply and chemical properties of the brain and nervous system, and then follows a truly admirable summary of the physiological relations and functions of the several parts of the brain and nervous system. Here the labours of Flourens, Laycock, Luys, Van der Kolk, Goltz, Hughlings-Jackson, Hitzig, Ferrier, Purkinje, Longet, McKendrick, Robertson, and many other observers and investigators are epitomised and digested with great ability and discrimination, and all that has been established with regard to localisation is clearly brought out. From physiology Dr. Aitken passes to morbid anatomy, and discusses the pathological relations of the several parts of the brain and nervous system, furnishing guides to the clinical investigation and diagnosis of disease. The laws of isolated conduction of sympathy and irradiation and of mental manifestations are explained, and the localisation of disease in the brain, spinal cord

and nerve trunks, is set forth as explicitly as our present state of knowledge warrants. Passing from general and preliminary considerations to the concrete and actual examination of disease, Dr. Aitken introduces first functional disorders, and secondly substantive diseases of the brain and nervous system. Neuralgia, cephalalgia, anæsthesia, labyrinthine vertigo, infantile and puerperal convulsions, scrivener's palsy, largysmus stridulus, paralysis agitans, chorea, hysteria, rhythmical chorea, epilepsy, catalepsy, tetanus, hydrophobia, sunstroke, general paralysis, hemiplegia, bulbar paralysis, paraplegia, spinal hemiplegia, polio-myelitis, progressive muscular atrophy, pseudo-hypertrophic paralysis, locomotor ataxy, local palsies, delirium, pachymeningitis, cerebral and spinal meningitis, tubercular meningitis, chronic hydrocephalus, encephalic apoplexy, softening of the brain, encephalic tumours, disseminated sclerosis, aphasia and insanity are in turn and exhaustively considered, besides many subsidiary topics which we have not enumerated. Each disease is dealt with in all its bearings, in a scholarly, systematic, and practical way, a definition being attempted in each, and all that is known of symptomatology and pathology being ably expounded. The sections on epilepsy and insanity are the most elaborate, and mount indeed to the merit of tolerably complete treatises on those subjects. The medico-legal relations of insanity are not overlooked, nor are the relations of states of the eye to diseases of the nervous system, a chapter being added on that topic.

If we were to find any fault with Dr. Aitken's 'Science and Practice of Medicine' it would be to the effect that the directions for treatment which it supplies are not always as full and minute as could be desired. As medicine advances and becomes more scientific—and that it is doing so Dr. Aitken's book is a most signal proof—treatment emerges from empirical simplicity into rational complexity. The means used become more simple—prodigious concoctions giving place to single remedies—but the considerations affecting the use of these means become infinitely more intricate and numerous. Determined and regulated no longer by an all-embracing theory, a name or a rule of thumb, but by a better appreciation of the causes of disease and a juster

recognition of the disordered processes and material changes which form its constituents, treatment has branched out into a manifold heterogeneity, requiring a minuteness of description that was formerly unnecessary and unknown. Enlarged by the discovery of new remedies and by the new applications of old ones, and modified from time to time in each case in accordance with the variations of symptoms which modern observation keeps under close and incessant supervision, the treatment of disease has become laborious, and demands in many instances a minute observance and resource, scarcely inferior to what is required in Listerism as applied to surgical wounds. Of course Dr. Aitken is alive to all this, and has under many diseases entered fully and explicitly into the whole duty of the medical attendant, from first to last, tracing out the parallelism which should be observed between treatment and the progress of morbid processes, and supplying prescriptions which have been well selected, and must prove eminently useful to the busy practitioner. Nothing could be better than the directions he gives for the treatment of megrim, of epilepsy, of tetanus, and of neuralgia. But under other headings his account of treatment and recommendations regarding it are occasionally somewhat meagre. Thus the treatment of catalepsy is dismissed in half-a-dozen lines; that of locomotor ataxia in half a page, and that of insanity, which is considered as a whole and not in relation to the many distinct varieties of mental aberration, which require distinct courses of medical and moral management, in five pages. In a few cases, too, we find no mention made of remedies which are believed to be of real value. Thus nitrite of amyl is not alluded to under the treatment of angina pectoris, nor Calabar bean under that of general paralysis, nor hyoscyamine, conium, chloral, nor arsenic, under that of insanity. Such omissions, however, are few and far between, and can only be discovered by careful search, and it would be ungracious to refer to them save with the desire that every flaw, however insignificant, should be removed from a classical work, which does honour to British Medicine, and is a compendium of sound knowledge.

J. Crichton-Browne, M.D.

Clinical Cases.

A CASE OF EPILEPSY:—AURA OF TRUE VERTIGO — EMOTIONS OF ANGER AND REMORSE — VAGUE COMPLEX REMINISCENCES — RIGHT-SIDED SPASM—FAMILY HISTORY.

BY CHARLES MERCIER, M.B. (LOND.), F.R.C.S.

Senior Assistant Medical Officer at Leavesden Asylum.

Mary H., æt. 36, unmarried. Family history from patient, who is fairly intelligent. Her mother used to have fits, in which she screamed, fell, and used to lay hold of things. On one occasion she fell into the fire and took hold of the bars, burning her hands severely. Mary H. had two sisters, of whom the elder had severe fits, in one of which she died. The other had fits until puberty, at which time they ceased. This second sister was badly frightened one day, was brought home, and died the same evening, but whether in a fit or not the patient cannot say. The mother had no brothers or sisters. The father died "asthmatical."

In obtaining the following account, the utmost caution was exercised to put no questions that should in the least degree suggest the answer, and the description of her sensations is taken down as nearly as possible in her own words.

She has both "fits" and "sensations," which both begin in the same way. They come on with a giddiness. She feels very trembling, and the place and everything seems as if it were really going round before her. Subsequently she says that everything seems to be dancing and going all manner of ways; but on being told to move her hand in the direction in which things appear to go, she waves her right hand with a circular motion from left to right, and adheres to this description when severely cross-examined. This continues for a "good while," and then is followed by a feeling "as if she must cry out," "as if she must give a good cry and hold on to some one." This is because she feels the fit coming on, and wants to hold on to people, so that they can take care of her

and see that she doesn't fall, and doesn't do anything wrong. On further questioning she says that she catches hold of people because her head feels queer, and because she is afraid of doing anything wrong. She repeats many times in the course of her examination that she hopes she does nothing wrong in the fits, that she doesn't mean to do anything wrong, and that as soon as the fit is over she always asks what she has done, and feels very sorry for having done wrong and for having said anything that she ought not to say. As a fact, however, she neither uses bad language nor is she violent during the attack, nor does she ever express regret after it, as she imagines that she does. Besides this she says, "Before the fits I think very much of where I have been (long ago) and how they might be, and I should like to be with them again, all manner of things." Again, "I feel as if I should like to go to them, and say to them, 'I should like to do my duty to you and work with you, and be happy with you.'" The "they" refers to her sister and her sister's family, with whom she once lived. These feelings occur after the giddiness, before the "fit," but at the end of the "sensations." To this statement she adheres without wavering when questioned on different days. Before the "sensations" she feels "workified." "As if I wanted to be working around and around and doing things." *After* the fits she has the same feeling. Again, she says, after the fits she feels as if she were going to hit people. She feels towards them as if they had done something at her. She feels that she would like to hit them in order to vent her spite on them—to be revenged on them. These feelings towards other people she does not experience at all in the sensations.

While examining this patient, I was called away to observe another who was in a fit at about six yards' distance. While observing this second patient my attention was attracted by a harsh cry as of some one in terror, and turning round I saw Mary H. sitting in the chair which I had just quitted and still emitting the cry. I immediately went to her, and on my approach she rose from the chair and stood beside it, with a grieved and anxious expression of face. The whole affair passed so quickly that it is not possible to particularise further than to say that this is the impression that her face gave me. It is certain that her "grief muscles" were strongly in action. On immediately asking the nurse who was standing beside her during the attack to which side her head was turned at the outset of the fit, she answered, "To the right"; and it may therefore be taken for certain that this was correct. On then at once asking the patient how she felt, she answered, "I feel better now. I feel as if I want to go to work—

to be doing something." She also said in answer to questions that she knew she had just had a fit. On the following day, however, she was ignorant of the whole affair, and of my asking her about it. The duration was between five and ten seconds.

Nurse's Report.—Mary H. has both "sensations" and "fits." The "sensations" are very frequent—one or two every day. The first thing noticed is that she gives a slight scream and makes for a chair. There is generally time to get her into one, and if she did not reach one she would fall. She "works about," and has to be held (to prevent her from falling) for a few minutes (probably moments). Then she becomes very restless and begins to undress herself. The nurse who has witnessed these "sensations" daily for four years, has never known her catch hold of people either in or after the "sensations," neither has the patient ever attacked people or been known to look angry. As soon as the restless movements cease she gets up and goes to work.

The "fits" come on sufficiently deliberately for the fall to be prevented, or she would fall. Judged by the standard of the other ninety-nine epileptics under the charge of this nurse, the fits are rather severe; they affect both sides, and are followed by "deep sleep," and as soon as that is over she gets up and goes to work. These severer attacks occur at intervals of three or four weeks. At longer intervals—of three or four months—she has batches of fits, after which she is alienated for a short time, strips herself naked, and runs up and down the ward screaming.

Remarks.—The importance of this case in regard to the speculations of Dr. Hughlings-Jackson in the last number of 'BRAIN' is manifest. The under-mentioned are the points on which it bears directly. The epileptic attack, beginning with true vertigo, is associated with right-sided spasm, and objects appear to be displaced to the right. This is in harmony with Dr. Jackson's supposition. 2nd. There was after the fit no sign of aphasia of any degree, i. e. no using of wrong words or approximate expressions even. 3rd. The vague complex reminiscence occurred in association with right-sided spasm at the outset. 4th. This intellectual state occurred in association with "the objective warning of vertigo." In the last three particulars the occurrences in this case are not in harmony with Dr. Jackson's speculations. It should be added that the patient is right-handed.

The case is important also as illustrating other points in the natural history of Epilepsy.

The identity of the feelings experienced during the "sensation" with those experienced at the outset of the "fit" is

proof, if proof were needed, of the identity of nature between *petit mal* and *grand mal* with universal convulsion. It shows that the discharge in the latter begins in the same region and spreads in the same manner as in the former, and that the difference between the two forms of seizure depends mainly upon the greater or less extension of the discharge. Whether the rather elaborate mental states occur during and in consequence of the discharge itself, or whether they occur from over-activity of regions from which control is removed in consequence of the discharge in regions above them, is immaterial to the present issue. Whatever the *modus*, the same physical state must accompany the same mental state.

Another important fact is that what she calls the "workified feeling," "as if I wanted to be working around and around, and doing something," occurs before the sensation and after both the sensation and the fit. In other words, the same mental state, and therefore the same physical process in the brain, occurs early in the onset and late in the recovery, corroborating and illustrating the doctrine that the regions of the brain resume their functions in the inverse order of that in which they were discharged; and that this law obtains as truly in those higher and less deeply organised regions of the brain whose disablement corresponds with loss of consciousness, as in those lower and more deeply organised regions whose disablement corresponds with hemiplegia.

ON HEMIPLEGIA AND HEMIANÆSTHESIA IN AN IDIOT BOY, AS THE RESULT OF PARALYSIS OF THE LEFT CEREBRAL HEMISPHERE, FOLLOWING A BLOW ON THE HEAD.

BY DONALD FRASER, M.D., PAISLEY,

Visiting Physician, Riccartsbar Asylum.

So far as my experience and reading are concerned, the case which I am about to relate is, in some respects, unique. But for the opportunity of making a post-mortem examination, the symptoms would have been considered as indicating plainly some such organic lesion as a considerable effusion of blood affecting the left hemisphere; while as it was, the most careful examination failed to show any gross lesion. The case has further some special interest at present, when so much attention is being paid to the subject of hemianæsthesia.

W. C., aged 11½ years, of illegitimate birth and the subject of congenital idiocy, was admitted into Riccartsbar Asylum, on Jan. 20th, 1878, in good bodily health. He was a chubby-faced active little fellow, about 4 ft. in height. He walked with a very slight inclination forwards, and carried his right arm awkwardly, or as if he had not quite the same control over it as he had over his left; and when he was allowed to feed himself, which was not often, he used his left hand. He was readily attracted by a bright object, and was often troublesome in his attempts to examine such objects as one's watch guard. His intelligence was very low. His speech was confined to a few words, used apparently without reference to their meaning. These words consisted of names which he must have been in the habit of constantly hearing before his admission to the asylum. "Granny," for instance, was often uttered by him. Sight and hearing were apparently normal. He was wet and dirty in his habits. His right testicle was absent.

His head measured in circumference	19⅜ inches
Antero-posterior diameter taken from root of nose to occipital prominence	12½ „
Distance from the one meatus to the other over vertex . . .	5⅜ „
Distance from the centre of forehead to meatus	5¼ „

He was reported to have had a severe illness about a year ago before his admission, which was marked by head symptoms, and preceded by a fit. It was also reported by his friends that he was in the habit of taking a "fit" about once a year or so, though during his stay of ten months in the asylum he was free from epilepsy.

On the 30th Nov. 1878, he was thrown down by one of his fellow patients, so that his head struck the floor of the day room with some force, though it was not sufficient to bruise the scalp, or produce any other external mark of injury. He appeared to be none the worse immediately after the accident, but in about 15 or 20 minutes was found by the head male attendant lying on the floor, in what he, the attendant, described as a fit, which was attended by pallor of the face and vomiting. A few hours later he was unconscious. His condition next morning, twenty-four hours after the accident, was as follows: His head and eyes were turned to the left side, his right eye most so. The pupils were dilated, but reacted to light. The whole of the right side of the body, including the right side of the face, was anæsthetic, while the right arm and leg were paralysed. Tickling the sole of the right foot caused a scarcely perceptible reflex movement. A pin thrust into various parts of the right arm and leg was followed by no action whatever, but when applied to the left caused shrinking and withdrawal of the limb. The temperature in the axilla was 102°. Respirations were 18, and pulse was 84. His bowels were opened freely during the day by means of an enema. Beef tea and milk, when given to him, were swallowed easily. Next day, the 2nd Dec., his pulse rose to 90, but the breathing continued quiet and regular; egg and milk mixture was given, and swallowed without difficulty. The paralysis of sensation and motion on the right side was still complete, otherwise he lay like one in a profound sleep. On the third a blister was applied to the vertex. 5th Dec., pulse 90, respiration 16. The conjunctivæ of both eyes were injected, the right particularly so, while the lower third or more of its cornea was muddy and soft-looking. 6th Dec., he was so far conscious that he picked up with his left hand the tobacco-box of a fellow patient when it was put within his reach. He now presented a very striking appearance. His right limbs were limp and motionless, the paralysis of motion

being still profound; the cornea of the right eye, so far as it was uncovered with the lid, was white and ulcerated. The breathing was quiet and regular. His consciousness was so far restored that on his attention being called to the tobacco-box which was held near his left hand, he slowly and in a dreamy way grasped it, and kept hold of it. Pricking the left arm and leg was followed by expressions of pain and movements of the limb. This improvement did not continue, for on the next day his pulse rose to 120°; his temperature being 98½°. The tobacco-box now required to be put into his hand, which closed over it slowly as that of a sleeping child might do. He still breathed quietly and regularly as if in a deep sleep.

On the 9th Dec. pulse 140, respirations 20. The left cornea now appeared softened in its lower third.

10th Dec. The left side was now found to be hemiplegic, and he was evidently sinking. He died next day at 11 A.M.

A post-mortem examination of the body was made about 30 hours after death by Dr. Richmond Paisley and myself. We were assisted by Dr. Cunningham, now of Alva, who had seen the patient with me during life. There were no external marks of injury on the body. The scalp and skull were carefully examined, but shewed no evidence of injury. The thoracic and abdominal organs presented no appearance calling for remark. The calvarium was of medium thickness, and the dura mater was slightly adherent to it in several places. While the brain was still in situ the left hemisphere was seen to be fuller or larger than the right. The superficial vessels of the dura-mater were full and somewhat hyperæmic. The subarachnoid fluid was rather more abundant than usual. On removal of the brain the left parietal lobe was seen to be distinctly larger than the right. There was at the same time a marked depression of the orbital surface of the left frontal lobe. The optic thalamus and corpus striatum on the left side were also unmistakeably larger than those on the right. A desire to preserve the relation of the different parts prevented me from weighing them. Very careful incisions were made, particularly in the region of the left optic thalamus and internal capsule, but no lesion of any kind could be discovered. The convolutions appeared normal in arrangement and number and were deep, while the grey matter seemed deeper than usual. The brain weighed as a whole 32¾ oz. It was set aside, the relations of the various parts being carefully maintained, and submitted to a second examination by Dr. Joseph Coats, Pathologist to the Western Infirmary, Glasgow, who was also unable to detect any lesion of it or its membranes.

The spinal cord was examined and found normal, and por-

tions of it were also hardened in chromic acid. Sections presented nothing unusual when examined microscopically.

So far as the weight of this brain was concerned, it was only a few ounces below the average weight of the brain at his age. The obvious departure from the normal condition was the unequal size of the hemispheres and central ganglia. The greater size of the left hemisphere, or more strictly the left parietal lobe, was not here, as is often or generally the case where such a symmetry is found, due to atrophy of the right, for the latter was functionally the more perfect of the two. Dr. Crichton-Browne[1] refers to a pronounced excess in weight of the left over the right parietal lobe in both sexes, amounting in men to 8·1 grammes, and in women to 4·8 grammes, and considers this difference to be due to the higher development and education of the left hemisphere, which presides over the more specialised and voluntary motor performances of the right side of the body. In the brain under consideration, the excess in size and presumably in weight of the left parietal lobe over the right was so considerable that it at once caught the eye; yet the right side of the body was here the least capable of voluntary motor performances.

Mere inequality in size of the two hemispheres is of itself, then, compatible with the vigorous and normal performance of the cerebral functions. Yet, according to Griesinger, inequality of the hemispheres is frequently observed in the brains of idiots, where, however, it is obviously associated with pathological conditions. The apparently greater depth of the cortical layer in this brain was in keeping with another peculiarity referred to by Griesinger,[2] that of an unusual abundance of grey substance in the brains of some idiots. He even refers to the grey matter in the brains of certain idiots as being "greater than the white." The most likely explanation of the nature of this condition, particularly where the excess is so great as that referred to by Griesinger, is that the brains in question were in an undeveloped condition; for, according to Meynert,[3] in the cerebral medulla of "the fully developed brain no finely granular substance appears to be present, but during the period of development it exists, and probably occasions the *more grey than medullary aspect of the immature organ.*"

Only a complete microscopical examination of the component parts of such a brain as we are discussing would be of any great value. Apart from this, I examined numerous

[1] 'On the Weight of the Brain and its component parts in the insane.' Brain, Vol. II. p. 65.

[2] Griesinger 'On Mental Disease,' p. 360.

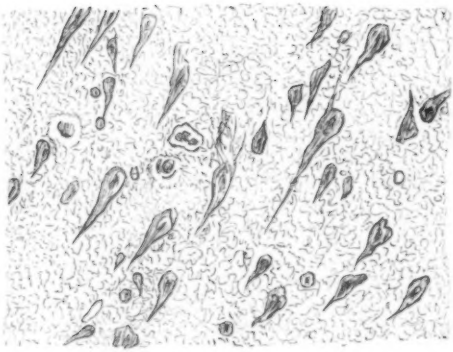
[3] The 'Brain of Mammals,' by Theo. Meynert, p. 390. Human and Comp. Anat., New Syd. Soc. Trans.

sections, made chiefly from pieces of the left cerebral hemisphere. I have satisfied myself, as the result of repeated examination, that the pyramidal cells, particularly of the second, and to some extent of the third, layer of the cortex indicate a condition of defective development. The processes are fewer than normal; indeed, the apical process is, in most instances, the only one present. The cells are, as a whole, more ovoid in shape than is usual, and many have what might be described as a somewhat withered appearance. It is necessary to speak with some degree of caution on this point when dealing with hardened specimens. To some extent they approached in character to the description which Dr. Bevan Lewis, in 'BRAIN,' Vol. II. p. 364, gives of the appearance of these structures in such epileptic idiots' brains as he had examined in the fresh state, though much of what he describes could only be observed in that state. The sketches *a* and *b*, magnified about 300 diameters, made with the aid of the camera lucida, give a very accurate representation of a specimen where, if anything, the appearances were more normal than in many others.

a represents a group of well-marked large pyramidal cells of the inner portion of the third layer, specially selected because of their large size and normal appearance. They also serve to show, in contrast to *b*, that the angular or pyramidal shape is not necessarily affected by the hardening agent. In *b* the general appearance of the larger cells of the second layer is well represented.

There can be little doubt that in this case the hemiplegia and hemianæsthesia were due to changes affecting the cortex of a large portion, if not the whole, of the left hemisphere, and influencing more particularly its cells. This boy having an unstable and unsymmetrical brain, with at least its cells imperfectly developed, this condition being most marked in the left hemisphere, suffers as above described from the effects of a blow on the head: a blow which in a healthy child might have given rise to the symptoms of slight shock; or at most to what I have occasionally seen from such a fall, a slight epileptiform seizure, succeeded immediately by involuntary emission of urine and a sleep from which the child awakes well. Here indeed the paralysism was indeed preceded by a fit, but instead of passing off it continued, and was also more profound than is usually seen in post-epileptic paralyses. So much was this the case that, as we have seen, ulceration of the lower half of the right cornea took place, the portion, by the way, which was most exposed to external causes of irritation. It was only during the last two days of his life that the right hemisphere began to indicate an equally serious failure

a.



b.

To face p. 510.

in its functions. During the first few days it showed signs of slight irritation. At least to this I partly attribute the forcible turning away of the face and eyes from the paralysed side, although no doubt the usual explanation of this symptom—that it is due to the muscles of the sound side being unopposed—is largely the true one.

It is probable that a similar train of symptoms might have come on at any time, even as the result of comparatively slight irritation. The serious illness which he had a year before his admission to the Asylum was post-epileptic, and must have been, to some extent, of the same kind as above described.

There is no reason to suppose that the usual vascular changes which take place in the brain as the result of slight or moderately severe shock were absent here. The difficulty is to understand how, in the absence of any apparent inflammatory changes, the paralysis should have persisted, though on the sixth day there was an attempt at recovery, as evidenced by the return to a dreamy sort of consciousness; yet it is to be noted that on that day the loss of motion and sensation on the right side was as marked as before. It is possible, though it is purely a conjecture, that the relapse which occurred next day was due to an unobserved epileptic discharge. Essentially the fault appears to have been a molecular one, and the persistence of the disease was not due to vascular changes so much as to the continued inability of the brain-cells to appropriate such nourishment as was necessary for their activity. In some respects it is allied to cases of functional hemiplegia and hemianæsthesia. Dr. Wilks, in his lectures on 'Diseases of the Nervous System,' says that, in his experience, a perfect hemianæsthesia has been always functional. The anæsthesia of the hypnotic state to which those strange cases of hysterical anæsthesia, of which we have been hearing so much of late, are closely related, presents us with numerous illustrations of temporary loss of function of certain portions of the brain. Such phenomena occur chiefly, if not entirely, in those whose nervous centres are unstable and easily exhausted, and whose power of inhibiting lower centres by higher is very feeble. In such persons any slight and continued irritation, whether it come from the ovary or from the operations of the mesmerist, soon exhaust the centres to which the irritation is propagated, exhaustion rather than increased tension being the condition to which certain groups of cells are reduced. In the light of such considerations and of the imperfect development of this boy's brain, what at first was a very puzzling case becomes comparatively clear.

AN OBSCURE CASE OF BRAIN DISEASE.

BY MONTAGU HANDFIELD JONES, L.R.C.P.

MENTAL derangement extending over six years; absolute deafness; lupoid disease of nose; death from catarrhal pneumonia; autopsy; hæmatoma; syphilitic disease of cerebral arteries.

The following case, which was seen at different times by several distinguished men in London (including Dr. Russell Reynolds, Dr. Sieveking, and Dr. Monro), and the autopsy of which I performed at my father's request, may perhaps be deemed worthy of perusal by the readers of this journal.

Mrs. B., æt. 49, *seen Feb. 6th*, 1874.

On the evening of December 1st, 1873, she was taken with violent giddiness and sickness, this latter symptom persisting for two days; she had not eaten anything that could have disagreed. Since that time she has suffered from "a wavering" in her head, and a weight and tightness at the vertex. She cannot endure music now, though she was an excellent musician. For the last year and a half she has been getting deaf. In 1851 she had a touch of brain fever. Has never had any children. Catamenia ceasing.

At present she is nervous and anxious; complains much of the wavering in her head; is very fidgety about trifles; the deafness is rather increasing; she complains of a sensation of grittiness in the eyes. She needs support in walking, or else she would seem "tipsy." On Feb. 19th she had a species of fit, in which she was unconscious for two or three seconds. Her hands tremble at times, especially the right when she holds it out. Throwing head backward relieves the giddiness. The fundi of her eyes are pale.

About end of March 1874 her condition is the following: No better. Urine pale, sp. gr. 1011, alkaline, not albuminous; she feels some sickness at times; is unable to read, as the letters all run together into up and down lines. She has to keep her feet well apart in walking, and to watch them; when walking she totters much, and seems afraid of falling

backwards. Has difficulty when following my finger to keep both her eyes fixed on the object, as one starts aside, when about 30° from median line. Left pupil slightly larger than right; she often sees double. Grasping power of both hands equal.

On *April 9th*, 1874, some difficulty of swallowing is reported, and fourteen days later this symptom is still present. On the 18th she was suddenly taken with ptosis of left eyelid, and much dimness of vision in both eyes. The left eye is the worst, and if it is partially open she has double and distorted vision; objects on the wall are all V's and A's, *i.e.* the boundary lines are displaced and angular. Her two eyes, as she has often observed, have different foci. She walks about still, but in a very unsteady manner.

April 28th.—Left internal rectus is much paralysed; globes do not move harmoniously. There is almost complete "*loss of speech*" to-day.

May 2nd.—Ptosis much less. Says she can't see, but does really, even if objects are very small. Is unable to work or read. Speaks a good deal, but is often at loss for a word; she uses absurd expressions, as "jenkins" for "pills." Mind seems weak, but she converses with her friends. By the 11th she has lost still more the memory of words, and cannot recall her friends' names. Mourns over her loss of sight, and says that she is going "blad," "blide" meaning to say "blind."

May 18th.—No material change; her morbid state seems to be largely mental, for while she goes on repeating continually "I can't see," and fretting that she cannot read or work, she undoubtedly sees to a considerable extent; this morning she called her husband's notice to two thrushes hopping about in the garden. Her hearing is decidedly improved, there is no ptosis; but the movements of the globes are not quite harmonious. No paralysis of the limbs. At times is violent; has difficulty in making persons understand what she wants.

May 21st.—Improving, but cannot read yet; this seems to be more from difficulty in conceiving the sounds of printed characters than in simply seeing them. She says that she cannot comprehend them, has not the sense, that she is mad. Shows me her collection of post-office stamps, but cannot remember or make out what country each belongs to. Laments that she cannot keep her account books. The defect is evidently in the intellectual centre chiefly; she says she feels strong, she is incessantly chattering, and displays a morbidly increased, though deteriorated, mental activity.

May 25th.—Says she knows everything and can do nothing. She knows what she wants done, but cannot express herself to say what it is. She uses wrong words at times, or words very

partially resembling those which she wishes to use. Gets quite impatient and excited, because she cannot read and work and manage her affairs. Walks well.

May 28th.—She has now had for about a fortnight pretty regularly alternating good and bad days; one day very excitable, petulant, complaining, and troublesome; the next much more cheerful, amenable, and rational. She can certainly see, but cannot read, or understand what is read to her. She certainly hears and walks materially better. She professes to forget names, even her own and her husband's; but it is evident that she can pronounce them with a little effort.

June 1st.—Continues to have good and bad days alternately, but the difference consists only in her being fretful, petulant, and desponding on the bad days and more contented on the good; there is no variation in the inability to read or understand what is read. Sees well, though she says she can't; she went to-day to her bird-cages, and remarked that the claws (very tiny ones) of one bird wanted cutting, but that she could not do it. There is this notable difference between her state and that of another patient I had, viz. that the latter could, though quite an old man, readily understand what was read to him, while Mrs. T. is quite unable to do so, though she can converse to a considerable extent. She says she is quite strong and knows everything, but somehow seems unable to use her knowledge. She missed a piece of work she had netted, and was quite in a temper about it; in enquiring for it she calls it a handkerchief.

June 15th.—Talks a good deal and uses many words, but says she can't talk, and replies to a question put to her, "I don't know what you mean." Shakes her clenched fist with vexation that she can't read. Her brain can evidently take cognizance of objects, and she has the memory to a considerable extent of words; but she cannot use language easily, and especially she has lost the faculty of apprehending the meaning of graphic characters, or of forming them, so that reading and writing are lost to her. She swallows now quite well. Ptosis gone. No paralysis anywhere.

July 2nd.—Seen by Dr. Russell Reynolds, who thinks there is some quasi-inflammatory process at the base of the brain, but no actively growing tumour. ℞ Hyd. Bichlor. gr. j. Pot. Iod. ʒ ij. Aq. ʒ iij. Capiat ʒ ij t. d.

Her state remains unaltered, she is melancholic, says that all food or drink is "blue, green, nasty poison," effervescing lemonade chokes her; she is querulous, morose, miserable. Said the other day, "Is it not a lamentable thing for me to be in this state?" The septum narium is perforated by an ulcer very near the columna nasi (lupus). She seems quite well in

bodily health; eats rather voraciously at night. Very morose and fretful, becomes very passionate at times, and when remonstrated with, says "that she was a lady once, but is not now." Declares she can't talk, can't hear, can't see, but does all three notwithstanding. She complains that she is inside and we are outside, meaning to express how she is cut off from the outside world by reading. She said to me, that I could not think how much she cried inside, meaning that she wept as it were in her mind, though there were no evident tears. She is very active, always fidgeting about, complaining that things are not in proper order; still retains her natural love for animals.

August 17*th*.—State much the same. She continues keenly alive to her loss of reading power, and frets much about it. The right ear is deaf, the left she can hear with. Her repertory of words and expressions is, her husband says, almost perfect, but she has forgotten her music almost as sadly as her reading.

Sept. 17*th*.—Aspect of features degraded as in insanity; same complaint that she cannot see or hear; says that any noise distresses her, goes through her head, yet she bore a short railway journey well. No power of reading or writing. Played cards one day, but complained that she played like a machine. Says she does talk such nonsense. Articulates perfectly, and has throughout.

Nov. 10*th*.—After a visit to Littlehampton she has improved greatly in memory and expression of thought and words; can write now a tolerable letter, but she cannot read yet. She knows however her husband's letters when they come, and is glad to have them, though she cannot read them.

June, 1875.—The lupus has spread slowly and eroded the outer part of the right nostril slightly, the septum is largely perforated. Her memory is good, she can play at cards, plays on the piano a little, but cannot read. Frets much, is decidedly inclined to be violent. Repeats again and again that she used to be a lady, to be so happy and clean, now is dirty, and can't bear to be washed. Says that her brain is broke, and that she can't walk.

March, 1876.—Had an attack three weeks ago, in which for a few days she quite lost her appetite, but had no fever or catarrh; since then she has been *stone deaf* in both ears. Also has become tottery on her legs; cannot walk up and downstairs, as she used. She sees perfectly; reads her name as I write it at the top of my prescription, she knows that latter is for her, says she won't take it. Memory good, eats and sleeps well; is violent and destructive, looks wild and haggard.

December, 1876.—Same condition, is stone deaf. Repeats

the old remarks that she was a lady, now is not: is dirty and hates being washed, throws herself about. No paralysis, walks upstairs in the dark, dresses herself quickly; can see very small things, reads letters, recollects events well and notices objects. Is always complaining of having had her teeth filed years ago. Nose in same state. Pulse not at all cordy.

December, 1877.—Has had an attack of jaundice for several days; mental state much the same; always harping on the old story of having had her teeth filed. Played a game of chess the other day.

January 3rd, 1880.—General health good. She reads now a good deal, reads commonly and understands written signs. Can only be communicated with by writing or signs, as she is stone deaf. She has no paralysis, throws herself about very much; is on her legs all day long. Always fretting about her teeth having been filed; says her nose is gone—all gone, and her teeth and her fingers near the tips. Plays chess well, dislikes being beaten. Is in many respects very rational; but Dr. Monro, who saw her at once, pronounced her insane, and offered to certify her. Says she cannot wash, is obstinate and very refractory, dislikes her nurse. Sleeps well. The sore which used to bleed and be scabbed at the right nostril has cicatrized after destroying a part of the septum and right ala. She dislikes now going outside the house. Is very fond of her husband, can't bear that he should be out of sight, but is more tractable in his absence.

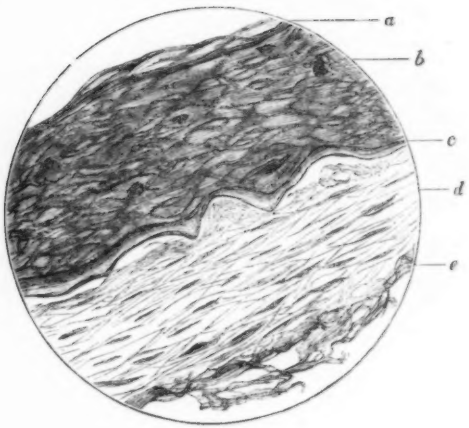
[*Treatment.*]—Iodide of potash and bichloride of mercury had both a fair trial, but without success; and as patient was very refractory the drugs were not forced.

July, 1880.—Died of broncho-pneumonia without any material change having shown itself in her recorded condition.

Post-Mortem of Mrs. B., October 1880. [Weather fine and dry.] Body emaciated; a good deal of hypostasis; scalp pale; a moderate amount of congestion of vessels of dura mater is present.

Dura mater carefully divided along level of sawn bone, and then the brain removed in the usual manner, though still covered by the dura mater.

On examining the surface of the brain, it was found that both hemispheres were covered by "a false membrane," which was adherent to the dura mater externally, and the cerebral arachnoid internally. Anteriorly this membrane extended under the frontal lobes as far as the lesser wings of the sphenoid bone, but in this position it became excessively delicate. Posteriorly it could just be traced to the internal occipital protuberance, while laterally it was just lost below the level of the horizontal limb of the Sylvian fissure. This membrane was



a. Epithelioid Lining.

b. New Growth.

c. Membrane of Henle.

d. Muscular Coat.

e. External Coat.

To face p. 547.

of a buff colour, homogeneous throughout in structure, firm and tough, showing on its internal surface at the parts where it was the thickest, peculiar stellate groups of injected vessels; the largest patch of these vessels was about the size of a halfpenny. By its outer surface the membrane was closely adherent to the dura mater, while between its inner surface and the cerebral arachnoid numerous well-marked vessels and what appeared to be fibrous bands were seen to be passing. The thickness of the membrane varied considerably—thus in the longitudinal median fissure, on the surface of the horizontal plate of the frontal bone, and laterally below the horizontal limb of the fissure of Sylvius it was a mere film; posteriorly over the occipital lobes, the angular, and the back part of the supra-marginal and superior parietal lobules, it was rather thicker than ordinary writing-paper; while at its thickest part over the three superior frontal convolutions, the ascending frontal and ascending parietal, and front part of the superior parietal and supra-marginal lobules it was fully an eighth of an inch in thickness.

The dura mater lining the cranial bones appeared natural; the vascular grooves on the bones were well marked. There was no thickening of the arachnoid. Along the margins of the longitudinal sinus (superior) small nodules of Pacchionian glands were very apparent.

The base of the brain, with the cerebellum, pons varolii and medulla oblongata, appeared normal. The arteries were large, gaping, and thick-walled; but there was no appearance of embolism in any of them.

On removing the false membrane the hemispheres were seen to be quite natural; the convolutions were not unduly flattened, nor abnormally adherent to each other. The auditory, facial, and other cranial nerves were quite natural: the two first-named lay normally in the meatus. On cutting up the brain the veins of the lateral ventricles were found rather large and full, but no tumour, undue softening, or other signs of morbid action could be traced in any part.

Microscopical Examination.—Sections from the hæmatoma showed that the latter presented all the features of a blood clot, which was becoming gradually organised. The vessels of the pia mater appeared to be perfectly healthy. Sections of the grey matter taken in varying situations were found quite normal. Examination of the large arteries at the base of the brain showed that important changes had taken place in these vessels. These changes which are represented in the adjoining sketch show clearly that the morbid process had its seat in the inner coat of the artery only; there was in fact a great proliferation and overgrowth of the connective tissue cells which

are normally found between the epithelioid lining and the fenestrated membrane of Henle. This chronic endarteritis has been fully described by Heubner in his admirable monograph on disease of the arteries of the brain, and is doubtless of syphilitic origin. The thickening of the coat of the artery varied at different parts of the wall; thus, while on one side of a section a considerable growth of connective tissue was apparent, at the opposite side frequently it could be made out that cell proliferation was only just commencing. In addition to this fibroid change it was clear that the disease tended to injure and impair the structure of the artery; thus, in many places the epithelioid lining was rough and ragged and projecting, at others the muscular layer appeared thinned and degenerated, while in other parts there seemed to be a disturbance of the connection and relation of the different coats one to the other.

In reading over the case reported above, two points seem brought prominently before us; firstly, that we have here an undoubted case of insanity, and secondly, that we are able to point to a well-marked and definite lesion as to the cause of the disease.

That insanity was present can scarcely be doubted, especially when such an eminent physician as Dr. Monro is willing to certify the case as such; and yet in some points the symptoms do not fully bear out such a statement. Thus the patient was undoubtedly at times melancholic and desponding, yet we never find that she regarded those around her with aversion or hate, as is so often the case; on the contrary, she retained her intense love for her husband and also her partiality to her pets quite unaltered. Apathy and desire for solitude are also frequently marked symptoms in cases of melancholia, but Mrs. B. always displayed considerable interest in her household matters, and never ceased lamenting that her infirmity prevented her from giving the needful care to these subjects. As regards solitude, moreover, the patient, though naturally confined almost entirely to her home, was yet always rambling over her own house, and seemed glad of the society of visitors when they came to call on her. Again, her consciousness of her own defects is painfully apparent. It is not that she has a morbid idea of a state which exists only in her own fancy; but she is dirty in her person, she is unable to attend to her house, she is disabled from music, reading, &c., and it is under a sense of these "real failings" that she suffers so acutely. Undoubtedly at times she was violent and refractory, but if we examine her actions at these times we fail to trace anything akin to maliciousness or distorted ideas; rather, the acts are those of a brain which is

irritated and upset from its true balance. Her own expression, so constantly used on these occasions, viz., "I can't help it," seems to point to the fact that the right sense was present, though some disturbing influence produced ever and anon an error or misdirection of cerebration. It seems important that we should recognise these points, because later on we shall find that these manifestations of disease are quite such as we should expect from the lesion present.

When we turn to the evidence afforded by the post-mortem examination, what we find is shortly this:—A large hæmatoma spread out symmetrically over both hemispheres, continuously but loosely adherent to the dura mater externally, more firmly but quite as universally attached to the arachnoid and pia mater below; in other words, over the grey matter of the convolutions, *i.e.*, over the substance which is without doubt the seat of the higher intellectual faculties, and probably in certain areas forms the centres for special mechanisms, we find superimposed a large irritating foreign body.

The symptoms of hæmatoma, as given by Aitken, Schuberg, and others, consist principally in general weakening of memory and intelligence, in giddiness and local head pains, in transitory fits of unconsciousness, in impairment of the power of speech, in excessive appetite, and lastly in paralysis of the face or limbs, the duration of this latter symptom varying greatly in different cases. While, however, we find that certain symptoms, as giddiness and weakness of intellect, are invariably present, it is evident, with regard to other symptoms, that many modifications or complications will arise, according as the extent, relations, and connections of the tumour vary. Regarding, then, in the present case the nature of the lesion, we can without much difficulty picture the character of the lesions likely to arise. Thus motor disorders will probably be present, and in the report we do notice that tottery walk, want of harmony in the action of the two eyes, ptosis of the left eyelid, and paralysis of the left internal rectus, were prominent symptoms. It is important to note that these paralyses came early in the disease and soon disappeared; for, as Goltz has shown in dealing with injuries to the convolutions, such temporary phenomena are due to the superficial lesions exercising inhibitory influences on the parts of the brain lying between the convolutions and the spinal cord, and not to any organic injury to these parts themselves. Again, since the false membrane was thickest and most adherent over those portions of the cerebral convolutions which are usually marked out as the centres for speech and sight, and therefore the irritation to these parts was likely to be considerable, we should expect to find that dis-

orders of speech and sight played a prominent part in the history of the disease; and such was actually the case. That mental disease should have shown itself markedly, is only what must have occurred under the circumstances. Dr. Wigan, in his work 'On the Duality of the Mind,' quotes a case in which a very slight depression of bone, caused by a tap on the head with a ruler, led to a complete change in the moral feelings of the patient; and if such a result could follow so slight a source of irritation, what wonder if the state of mind with which we are here concerned should have so prominently manifested itself! Moreover, in Dr. Wigan's case there was but one focus of irritation, and that limited; while in Mrs. B.'s case the whole mass of the convolutions was exposed to the fretting and irritation which must of necessity have arisen from their contact and connexion with the hæmatoma. The form too under which the mental derangement showed itself was striking. It is usual, in cases of hæmorrhage into the cavity of the arachnoid, to have intellectual disorders; but these usually appear in the shape of dulness and stupidity, somnolence and apathy, while in the present case quite the opposite state existed. There was, in fact, great mental activity, but it was of a morbid and deteriorated quality; the quick, bright habit of cerebration natural to the patient was not lost, but the brain was working under a cloud, was, in fact, working under some abnormal influence which misled and overpowered its normal healthy action. Her own complaint "that she was inside, and others outside," exactly hit off the state of the case; for between her own inner consciousness and her natural healthy relations with the external world disease had erected an impassable barrier—a barrier the existence of which she fully realised, but the influence and restraint of which she could not in any sense control. Regarding the deafness, which was so marked a symptom in the case, it is noteworthy that it had existed in a less degree before the patient's illness commenced, and that there is not sufficient evidence to connect it in any way with the intracranial lesion which was found. The case is of interest as showing that some forms of insanity can certainly be attributed to intracranial lesions, while at the same time it points to the fact that adverse conditions, which produce only a comparatively slight and temporary derangement of motor mechanisms, are quite sufficient to cause extensive and lasting injuries to the higher centres of intellect and volition.

While thus far we have attributed all the morbid symptoms to the existence of a large hæmatoma, we must not forget that the presence of lesion in the arteries of the brain may have played an important part in the causation of the disease. In

reading over Heubner's researches on syphilitic disease of the brain arteries, two points can scarcely fail to strike us, viz., firstly, that the morbid state of the vessels in the case under consideration is in reality the same as that which he describes and pictures; and secondly, that many of the symptoms which he considers diagnostic of syphilitic disease of the brain arteries were undoubtedly present in Mrs. B.'s case. Two extracts from his paper will illustrate these points. At page 224 he writes thus:—"The second principal group of symptoms of our disease next to those of hemiplegia (or the third group, if we look on the short-lasting attacks of unconsciousness which introduce the hemiplegias or come on without such, as the first) turns on the injury of the higher intellectual functions, on the damage of consciousness, and thus on the functions of the grey matter of the brain. The disturbances relative to this latter are not less frequent than the paralyses; indeed, we may even say that they exist more constantly; moreover, they are not absent at times when paralysis shows itself only in a significant manner." Again, at page 225 we find, "The characteristic of the universal brain disturbance is, in a word, the incompleteness, the imperfection, of the severe symptoms, *e.g.*, the injury of the consciousness without its complete removal, the giving up of absolute activity without removal of the impulse of the will, the appearance of delusions and delirium, but the sudden return at times to reason; it is a state of partial somnolence, partial wakefulness, partial dreaminess." Against the idea, however, that arterial disease played a prominent part in producing our patient's troubles must be brought the fact that no softening or other morbid change in the brain substance, such as we should expect if the blood supply of the nervous tissue was seriously interfered with, could be found at the post-mortem; on the contrary, there was every appearance of the cerebral circulation having been as full and active as it normally is. That disease of the cerebral vessels may have caused the hæmatoma, or even may have aided in the development of the symptoms present, is quite possible; but to imagine that the amount of arterial disease existing was sufficient *per se* to produce any serious deterioration of the cerebral functions seems scarcely borne out by the evidence before us.

Abstracts of British and Foreign Journals.

Magnan on Word Blindness. (*Gazette des Hôpitaux*, January 24, 1880.)—M. Magnan, in a communication to the Société de Biologie, relates two cases of aphasia complicated with a special phenomenon, to which he has given the above name.

One case was that of a man who was seized with right hemiplegia and aphasia after a fall. A month afterwards the patient recovered the power of speech, little by little; he understood spoken language; he wrote, either of his own accord or from dictation; but he was incapable of reading either print or manuscript, even when the latter had been written by himself; and he could not name letters inscribed upon a board.

The second patient presented similar symptoms. He recognised objects which were shown to him, but could not name them; could write words thought or heard, but could not comprehend or copy what was written. He had lost the notion of the value of gesticulations.

M. Magnan also cited a similar case, reported by M. Brouardel, where on post-mortem examination a hæmorrhagic centre was found at the posterior part of the fissure of Sylvius adjoining the pli courbé.

He explains the pathology as follows. The retinal image is impressed on a first centre, at the level of the quadrigeminal tubercles; that is, the reflex centre for the eye. From this centre it is impressed upon a second, situated about the pli courbé, or perhaps about the occipital convolutions. In the second centre it is perceived as a sensation and received by the attention and the memory; this being the psychic visual centre. The ideas to which its elaborations in this centre have given birth cannot be utilised for speech, unless the communications between it and the convolution of Broca are intact. If they are interrupted, the patient can still see, speak, and hear, but he cannot acquire through his eyes any new idea.

As there is no disease of the eye, and because it is owing to a purely psychic phenomenon, this affection might be better described as "Cerebral Word Blindness."

Delaunay on Memory.—M. Delaunay has made a communication on the memory to the Société de Biologie, the result of extended inquiries. His results are classed under the following heads:—

Species.—Memory is a prominent faculty among birds and among certain mammals—the horse, elephant, &c. For example, a horse often has a better recollection of routes and localities than his master.

Race.—Memory was very highly developed among ancient nations, evidenced by their long poems, handed down traditionally before the invention of writing. The inferior races have better memories than the superior—the Negroes, Chinese, Italians and Russians who learn foreign languages with extraordinary facility.

Sex.—The adult woman has a better memory than a man. Actresses learn their parts more quickly than actors.

Age.—Young people have better memory than old. The faculty attains its highest development at thirty years of age, and thereafter declines.

Constitution.—The feeble are more highly endowed with memory than the robust; provincials than Parisian, country-folk than townspeople, lawyers than medical men, ecclesiastics than the laity, musicians than other artists. At the normal schoo's, Val de Grâce, &c., the pupils who have the best memory are not the most intelligent.

Individual Organs.—The right cerebral hemisphere has more of the faculty of memory than the left. Lesions of the former affect the memory more than the other faculties; lesions of the latter leave memory intact. It is common to all the nervous system. The organs which preside over walking, swimming and flying are endowed with more stable memory than those of the senses; those of the senses than those of the emotions; those of the emotions than those of the intellect.

Physiology.—Memory is more active before than after a meal. Cerebral congestion interferes with this faculty. Education diminishes it, and the illiterate man has a better memory than he who can write. Morning is more favourable to memory than the evening; summer than winter; the south than the north.

Pathology.—The memory is in a state of exaltation in certain maladies, such as hysteria, and depressed by poisons, typhoid fever, &c.

In conclusion, memory being the gift of inferior races, women, adolescents, the feeble, the less intelligent, the right cerebral hemisphere is in *inverse ratio to the evolution*. Still more so it is in a certain measure in *inverse ratio to the nutrition*, because it is augmented by circumstances which diminish the nutrition.

M. Delaunay proposes to apply the same method to the study of all the faculties. He divides them into two great groups. The first, the inferior, including imitation, superstition, &c., attain their maximum in the case of inferior beings—women, children, old men, feeble and in the right brain. The second, the superior, including conception, comparison, invention, &c., are best seen in the superior races, white races, masculine sex, the adult, the strong and the left brain.

A. R. Urquhart, M.D.

Buzzard on Transfer-Phenomena in Epilepsy produced by Encircling Blisters.—In the course of this paper, which was read in the Medical Section of the Meeting of the British Medical Association at Cambridge during the past autumn, the author referred to four cases published by him in the 'Practitioner,' October, 1868, in which remarkable results followed the application of blisters (especially encircling blisters) to "limbs" which were the seat of marked epileptic aura. In one, a tickling in the left arm had always preceded the fit. After the application of a blister encircling this limb the tickling was transferred to the left leg. In another, characterised by a similar aura, the fits and the tickling ceased after the application of an encircling blister. In a third, a sense of numbness in the left wrist was transferred to the right wrist. A fourth was a woman in whom fits had always been preceded by cramp in the right hand, and who after the blister was affected with cramp in both hands before her fits. In one of these cases a subsequent autopsy showed cerebral tumour. Dr. Buzzard had recently applied encircling blisters to some other cases with the following results. In one where the fits (two years' duration) had always been preceded by cramp in the left foot and shaking and numbness in the left leg, after the application of an encircling blister to this limb it was the *right* leg which shook and was numbed. In another, tingling in the left arm was the symptom, and after blistering there was jerking of both arms and the left leg. In a third, the attacks were preceded by cramp of the left hand. The patient had had a severe fit two days before being seen. The left hand was quite powerless. An encircling blister

to the left arm was applied, and a week after the patient complained that though her left hand had next day regained its power the right hand had *ipso facto* become weak. The dynamometer showed a power of grasp of 40 k. with the left hand and only 18 with the right. The author's original observations had been made many years before those experiments upon hysterical hemi-anæsthesia in France, in which removal of the anæsthesia was found to be accompanied by its transfer to the other side. He urged that the phenomena pointed to a power of influencing in some way nervous centres by impressions upon the skin, and referred to some trials of a therapeutic character which he was basing upon the observations. His aim was in a case of aphasia to rouse into activity the posterior portion of the third frontal convolution of the *right* hemisphere by directing powerful impressions to adjacent grey matter by means of painful electrical currents applied to the tongue, mouth, and left arm.

J. Hughlings-Jackson.

Luederitz: Experiments on the Effect of Pressure on Motor and Sensory Nerves. (*Zeitschrift für Klinische Medicin*, Bd. ii., 1.)—In the introduction of his interesting paper, the author observes that a considerable number of cases have been reported in which peripherical mixed nerves were entirely cut in two without producing loss of sensation in the corresponding parts, the paralysis of the motor fibres of the nerve being at the same time complete. *Richet* noticed in a case of neurotomy of the median nerve, that when he happened to touch the peripherical end of the nerve the patient gave signs of violent pain. Evidently in this individual that part of the median nerve contained sensory fibres which were in connection with some other brachial nerves. A similar case of this "sensibilité récurrente" is reported by *Remak;* afterwards *Arloing* and *Tripier* proved by experiments on animals that many nerves contain sensory fibres derived from sensory nerves in the neighbourhood; these fibres run a little way in a centripetal direction, and then enter into the nerve to which they really belong.

These facts explain to a certain extent those well-known clinical cases in which we find complete motor paralysis without loss of sensation; but there are other cases which cannot be explained in this way, and therefore the author made a series of experiments on rabbits in order to ascertain whether the resistance to injury of sensory nerves is greater than that of motor nerves. He applied a

ligature to the sciatic nerve, and then stimulated with a faradic apparatus both the central and the peripherical part of the nerves at a few centimetres distance from the place where the ligature lay. The distance of the two coils, divided in millimètres, expressed exactly the intensity of the current which was wanted for the production of a contraction of corresponding muscles, viz. of a sign of pain in the animal. In order to avoid the influence of "sensibilité récurrente" the crural nerves as well as all other sensory nerves of the leg were cut so that there was no sensory communication between the extremity and the brain but the sciatic nerve.

The result of these experiments was as follows:—The conductivity of motor fibres is invariably impaired earlier than that of the corresponding sensory fibres. It was found several times that 10–15 minutes after the application of the ligature sensibility and motility disappeared simultaneously, but much more frequently the author stated that the strongest faradic current applied above the ligature did not produce the slightest muscular contraction, whereas peripherical stimulation immediately afterwards made the animal display symptoms of pain. Sometimes motor conduction was completely interrupted, sensibility being at the same time absolutely unaltered. On the other hand, loss of sensation without motor paralysis was not observed in a single case. When the ligature was removed in cases of complete motor and sensory paralysis, in some cases both faculties were restored at the same time; in other experiments sensation appeared to have returned, while motor paralysis persisted.

The only way to explain these facts is to assume that sensory nerves have a greater resistance against traumatic influences than the motor nerve-fibres. As a practical consequence, the author points to Duchenne's observation: that the prognosis of traumatic paralysis is much more favourable when, the electro-muscular contractility being extinct, muscular sensibility is unaltered or but slightly diminished.

Brieger: A Case of Paralysis from Fright. (*Zeitschrift für Klinische Medicin*, Bd. ii., 1.)—The popular idea that violent mental shocks are capable of producing severe illness has not gained much ground in scientific researches. Only lately some cases have been reported by Kohts and Leyden, giving indubitable evidence of disease caused by intense fright (during the siege of Strasburg in the late Franco-German war). Similar ob-

servations have been made by Todd, Hine, Pasque, Berger, but all these were deficient with regard to anatomical examination. Leyden only has made autopsies in two cases of spinal paralysis: in the first case he found sclerosis of the middle part of the dorsal region of the cord; in the second case there was diffuse sclerosis of the upper and middle part of the cord in the dorsal region. The following observation, made at Professor Frerich's clinique, will therefore attract the attention of pathologists.

A prostitute girl, 23 years of age, without hereditary disposition to nervous diseases, going home late at night from a dance, ran against a man who was lying drunk on the stairs. She managed, however, not to fall, by clinging to the handle of a door, and there was certainly no definite injury to the spine. Trembling with terror she mounted with difficulty to her room in the third story and tumbled on her bed, almost fainting. After a little while she recovered from her fright, undressed and went quietly to bed, without feeling anything abnormal. At 4 o'clock in the morning she awoke, in consequence of intense pressure of urine, but found herself incapable of passing any water. At the same time she felt violent shivering. Hot bottles and tea applied by the landlady did her no good; she therefore went into the warm bed of the latter, in doing which the patient observed a considerable weakness in her legs. She then went to sleep; when she woke at 7 A.M. she found her legs entirely paralysed, and at the same time had a sensation of numbness and tingling. A few days afterwards she was brought to the hospital. There was now complete paraplegia, pain in the lumbar region when she tried to sit up, sensation of cold and heaviness in both legs. Mental functions normal, great depression of spirits. No fever. Residues of syphilis, for which she had been treated at the same hospital two years before this accident. Absolute anæsthesia of the lower part of the body up to the height of the second lumbar vertebra. Retentio urinæ. The treatment consisted in application of 4 grammes of mercury ointment pro die, and 2 grammes of iodide of potassium internally. No improvement in the following days. On the 12th day sloughing began in the sacral region, extending rapidly. A few days later, symptoms of spinal meningitis came on. Patient died on the 27th day after her reception at the hospital.

The *autopsy* revealed diffuse myelitis, extending from the upper lumbar region to the level of the 8th dorsal vertebra, chiefly localised in the posterior columns and the parts of the white

substance adjoining the anterior roots. Upwards there was found secondary degeneration of the postero-median columns (Goll's columns) reaching as far as the fourth ventricle of the brain.

The author thinks that in this case there can be no doubt as to the cause of the disease; he does not believe that the syphilitic history of the patient has anything to do with it, because there were no symptoms of visceral syphilis, and the other residues of the infection had been quickly improved by the anti-syphilitic treatment. He only admits that the patient's resistance against disease had been diminished by the venereal infection. Concerning the way in which mental shocks act on the cord, the author suggests that the fright brought on a sudden intense contraction of the blood-vessels, lasting for a considerable time and leading to necrotic destruction.

R. H. Pierson (Dresden).

Onimus on Modifications in the Excitability of Nerves and Muscles after Death. (*Journal de l'Anatomie et de la Physiologie*, 1880, No. 6.)—We know that after death muscles and nerves gradually lose their excitability. The vital functions are rapidly, almost instantaneously, lost as regards the brain, while in the spinal cord they persist for a short period, the grey matter losing its experimental excitability sooner than the white matter. With regard to the nerves, the loss of excitability does not, strictly speaking, occur in a regular order, commencing from central parts and gradually invading peripheral parts; the loss of excitability pervades the nerves of a limb as follows: first, the large cords; secondly, the filaments supplying extensor muscles; thirdly, the filaments supplying flexor muscles—a sequence that calls to mind common pathological events, e. g. dropped wrist. The author states that in the arm the radial nerve, in the leg the peroneal nerves are the first to lose their excitability.

The nerves of the ganglionic system preserve their excitability longest of all; the intestine has been seen to contract five or six hours after decapitation, and M. Robin elicited rhythmic pulsation of the right auricle many hours after death by distension of its walls. With regard to the muscles, their excitability during the first few minutes after death is enhanced; they are at their maximum excitability when the large trunks are inexcitable, their excitability is already on the decline when the smaller filaments are inexcitable. After a gradual death, which is an exhausting process, muscular farado-contractility is more rapidly lost than

after a sudden death; the decline is more rapid in birds than in mammals, least rapid in amphibians. Analogously with the event as regards nerves, extensor muscles lose their farado-contractility before flexor muscles (an hour or more). There is no appreciable difference between muscles of the superior and muscles of the inferior extremity. Muscles of the trunk and abdomen are the last to lose their farado-contractility. The author remarks that just as nerves which are most easily influenced by lesions during life, are the first to lose their excitability after death, so in muscles we find a similar correspondence; facial muscles going first, extensor muscles going before flexor muscles, muscles of the trunk going last. With regard to the efficacy of various stimuli, the greater energy of response to galvanism with diminished or absent response to faradism, commonly noted in pathological cases, is not observed as a post-mortem phenomenon, although very feeble galvanic shocks are competent when the strongest faradisation is ineffectual; also the contraction is tonic, persists during the passage of the current, and is not merely a spasm at its make and break. The above statements apply of course to percutaneous stimulation—we know in effect, that when with unbroken skin a muscle does not respond to faradism, it will still respond if the stimulus be applied to its bared surface; but the contraction now elicited is local, not general, and the author remarks that probably the stimulus does not act quâ electrical, but quâ mechanical, for at this period mechanical irritants are highly efficacious.

The approximate chronology of the events as regards muscle is as follows:—

2½ to 3 hrs. after death,		the muscles of the face and tongue, with the exception of the maseter,	have lost their farado-contractility.
3 to 4	„	the extensor muscles of the leg and arm, and the maseter	„
4 to 5	„	the flexor muscles of the leg and arm	„
5 to 6	„	the muscles of the trunk	„

With regard to the decline in nerves, the author states that of the large trunks excitability is lost at the end of 2 hours, that of the small branches flexor filaments retain their excitability often 4 hours longer than "the others" (extensor filaments? large trunks?).

The chronology of muscular galvano-contractility is not exactly laid down, the only statement on the point being that as faradism

loses efficacy, galvanism has a more marked action, and at a variable time after death remains alone efficacious; mechanical stimuli acquire a relatively greater action than electrical stimuli, and are finally the only stimuli capable of eliciting contraction; the last electro-muscular reaction which the author obtained was 9 hours after decapitation, being a local, sluggish, prolonged contraction to the direct application of the galvanic electrodes, apparently a result of chemical action.

As a sign of death, the author concurs in Rosenthal's opinion that electrical exploration is the best, most certain, and most rapid test of that event; the approximate time after death may even be pronounced on the above data. Thus, for instance, if in a limb faradism elicits response from the flexor muscles, no response from the extensor muscles, it may be affirmed that death has happened between 4 and 5 hours previously. If at the same time on the face we more easily elicit contraction by galvanism than by faradism, and if the contraction is sluggish, we are further assured that death has happened about 4 hours previously.

The electrical test fails only where it is necessary to know *rapidly* whether death has taken place, but the only case in which this necessity can arise is sudden death during pregnancy, where there is a possibility of saving a life by the Cæsarian section. In this single instance, other tests need be relied on, namely, the contrast between the appearance before and after sudden death, and the comparison of the maternal and fœtal pulse.

In sum, the electrical test affords the best sign of death, may inform us of the period of its occurrence, and is a powerful means of reawakening the circulatory and respiratory functions.

A. Waller.

Strümpell on the Pathology of the Spinal Cord.—In this paper (*Arch. f. Psych.* x. p. 676, and xi. p. 27), the author discusses the subjects of spastic spinal paralysis and combined systematic disease.

Spastic Spinal Paralysis.—The first case reported is that of a man, aged 25, who had contracted syphilis four years previously. Death happened within nine months of the appearance of the first symptoms. During the greater part of the time the symptoms were those characteristic of spastic spinal paralysis: there was gradually advancing paresis and paralysis of the inferior extremities, excess of the tendon reflexes, rigidity of the muscles, the usual spastic phenomena, and an absence of all sensory symptoms. It was not

however a typical case of the disease, for at an early period transient irritability of the bladder was noticed, and two or three weeks before death paralysis of the bladder and bedsore appeared, sensibility became affected, the muscles of the limbs atrophied, and the tonic contraction of the extensors was succeeded by contraction of the flexors.

The post-mortem revealed the presence of a diffuse chronic myelitis of the upper dorsal part of the cord. Westphal, it will be remembered, has shown that this part of the cord is very frequently affected in chronic myelitis. Though the disease was diffuse, it was observed that certain parts were more prone to be attacked than others, and that a striking symmetry obtained between the lesions on the two sides. Thus in the posterior columns, the columns of Goll were always diseased, and if the lesion spread to the adjoining columns of Burdach it was always the posterior parts that were most affected. In the lateral columns the posterior regions were more affected than the anterior, and the lateral limiting layers were almost intact. The anterior columns showed few signs of disease beyond a thin zone of marginal degeneration on each side. The changes in the grey substance were most marked in the anterior cornua; the most noteworthy appearances being the paucity of the ganglion-cells, the number of large spindle cells, the presence of cells with fatty granules, and, most important of all, the widely dilated vessels which gave quite a cavernous look to the tissues. In other places the adventitia of the vessels was much thickened. The myelitis had a further peculiarity; it did not consist of one large focus, but had a floccular or disseminated character, and presented a strong resemblance to the disseminated cerebro-spinal sclerosis. Indeed cases have been described in which the distinction was so puzzling that the diagnosis of chronic myelitis or disseminated sclerosis rested on the presence or absence of secondary degeneration.

In the latter disease secondary degeneration is not met with above and below the focus of myelitis; tracts of ascending and descending degeneration were respectively observed. There was ascending degeneration of Goll's columns and of the direct cerebellar tracts. The degenerated cerebellar tracts were traced upwards to the medulla. They lay nearer the olivary body than Flechsig figures them. There was descending degeneration of the lateral pyramidal tracts, of the direct cerebellar tracts, and for a short distance of a thin band of fibres on the outer side of each Goll's tract (bandelettes externes). The degeneration of the cerebellar tracts was found

as low as the upper part of the lumbar region. Clarke's columns were, throughout their length, considerably poorer in cells than usual. The connection between these cells and the cerebellar tracts seems much closer than that between the cells of the anterior cornua and the pyramidal tracts, for while disease of Clarke's cells very frequently accompanies degeneration of the cerebellar tracts, we often have disease of the pyramidal tracts without alteration of the cornual cells. How shall we explain this descending degeneration of the cerebellar tracts? Three hypotheses seem open to us. The morbid process may have spread from the pyramidal strands to the contiguous cerebellar tracts, or the case may have been one of systematic disease of the cerebellar tracts complicated with dorsal myelitis, or lastly, it may be that focal lesions of the cord in certain circumstances are associated with descending as well as ascending degeneration of the cerebellar tracts. The last view is adopted by Strümpell.

The slight implication of sensibility is remarkable, considering the extent of the myelitis. As the lateral limiting layers were intact, the author suggests that perhaps they are the conductors of the sensory impressions.

The next case was, as regards its symptomatology, a typical case of spastic spinal disease. The most remarkable abnormality was the excessive dilatation of the central canal of the cord. The dilatation commenced between the origin of the first and second cervical nerves and extended throughout the cord. It was greatest in the dorsal and upper cervical regions. In some places the cavity measured 8 mm. from side to side. In the upper part of the dorsal cord the central canal appeared double on transverse section, owing to a small diverticulum from the canal at a lower level. The dilatation was not the result of softening or shrinking in the surrounding tissue, but was a primary change due to abnormality of development.

Besides the hydromyelus there was degeneration of the pyramidal strands of the lateral columns. The appearances were exactly similar to those found in secondary descending degeneration, but as there was no evidence of direct interference with the tracts, and as the lateral limiting layers, which pressure radiating from the central canal would naturally affect most, were almost intact, as too the degeneration was not confined to the pyramidal tracts, but implicated also the cerebellar strands, Strümpell inclines to think that the case was one of hydromyelus accompanied by primary systematic disease of the pyramidal and cerebellar

tracts. A narrow belt of degeneration, probably the result of an extension of the disease from the central canal, surrounded the grey matter in the anterior and lateral columns. In the posterior columns there was no sign of systematic disease; on the contrary, the affection had an exquisite disseminated character.

Taking a general view of the case, the author remarks that there was a hereditary predisposition to nervous disease, which showed itself in the earlier stages of development in the anomalous condition of the central canal. Probably the faulty laying down of the strands of the cord, some systems being more affected than others, predisposed to the early appearance of the systematic affection.

Spastic spinal paralysis occurs in widely different diseases. We have considered at length two conditions in which it was seen, and to these Strümpell adds, giving cases, injuries of the spine (myelitis from compression), tumours of the cord, multiple cerebro-spinal sclerosis, chronic hydrocephalus, and those affections of the nervous system which are sometimes found as the sequelæ of acute illnesses, and especially of typhoid fever. It is impossible as yet to specify the anatomical lesion that underlies the disease, though, as Erb stated, primary systematic degeneration of the lateral columns often induces it.

In pursuing this investigation we must be careful to distinguish between the paralysis and the spastic phenomena. We may have the latter without a trace of the former. We must also remember the complexity of the group of spastic symptoms. This includes, besides the phenomena dependent on excess of the tendon reflexes, various kinds of muscular contraction and contracture, e. g., the contracture due to direct motor irritation (flexion of the leg with convulsive dorsal flexion of the foot, *Beugecontractur*), and the contracture due to reflex influences (extension of the leg with strong plantar flexion of the foot, *Streckcontractur*); and to these must be added localised convulsive movements that are produced by a variety of causes.

Combined Systematic Diseases of the Cord.—The first case presented the group of symptoms found in amyotrophic lateral sclerosis: paresis of the extremities, trophic changes, and spastic symptoms. There was simple atrophy of the muscles of the forearm and hand, but in the triceps and the muscles of the lower extremities, there was, in addition, a striking degree of fatty infiltration of the muscles: the reaction of degeneration was not observed. The most constant of the spastic phenomena was the

strong dorsal flexion of the foot and toes, with which was often combined flexure of the thigh and leg, making up what the Germans call a *Beugecontractur*. The tendon reflexes were excessive. Convulsive flexing movements were observed in the legs from time to time, and generally affected them alternately. The passive contractures observed in paralysed limbs must be distinguished from the spastic contractures just mentioned. The former are permanent, and the deformity of the limbs gradually grows worse. The latter vary in intensity at different times, partly owing to the varying intensity of the central irritation, partly to the varying irritability of the nerves and muscles. In the former there is club-foot; in the latter, dorsal flexion of the foot. Strümpell thinks that the spastic contracture is due to primary disease of the pyramidal lateral columns, the fibres being still in connection with their trophic centres, and hence retaining their irritability. Applying the same reasoning, secondary degeneration of the lateral columns cannot account for the contracture of hemiplegia, for the fibres are separated from their trophic centres, and speedily lose their irritability.

A peculiar feature of the case was the association of movements in the lower limbs. There was not only an abnormal irradiation of the voluntary impulse to other muscles on the same side, but the patient could never flex his thigh either voluntarily or as the result of a reflex excitation, without inducing very intense dorsal flexion of both feet. There was complete incontinence of urine, and ultimately purulent cysto-pyelitis. Sensibility to cutaneous stimuli was perfect. A feeling of cold and formication was experienced in the lower limbs, but never to any great degree. Towards the end, pains of some severity were felt, but they were quite different from lightning pains.

The case was diagnosed as amyotrophic lateral sclerosis, though there were evidently important points of difference between it and Charcot's cases of this disease. For example, in Charcot's cases the disease generally commenced in the upper extremities, and travelled downwards; in this case an exactly opposite course was pursued. Further, in Charcot's cases there was usually atrophy, without fatty infiltration, of all the muscles of both extremities, and there was no paralysis of the bladder. Nevertheless, there seemed good ground to expect systematic disease of the pyramidal strands, and lesion of the anterior cornua.

The lesions found on *post-mortem* examination were (1) complete degeneration of the lateral pyramidal tracts of both sides in their

whole length, and of the anterior pyramidal tract of the right side down to the lower dorsal region; (2) degeneration less intense, of the cerebellar tracts; and (3) degeneration of the posterior columns. The anterior cornua were quite healthy. The cells of Clarke's columns were abnormally few in number in the lower dorsal region. The disease nowhere extended into the medulla oblongata.

The distribution of the disease in the posterior columns was remarkable. The condition of each column was as follows: (1) a narrow belt of normal tissue bordering the posterior fissure; (2) external to this, the degenerated Goll's column, triangular in shape; (3) external to this again, the degenerated 'postero-external region of the posterior column,' also triangular in shape; (4) in the anterior part of the column, and with its apex directed back between the apices of (2) and (3), a third triangular area, of normal tissue; (5) in the mesial line immediately behind the posterior commissure, and at the apex of Goll's column, a small band of degeneration; and lastly (6) a narrow intact strip on the inner side of the posterior roots. The symmetry of the disease on the two sides, and the occurrence of the same sharply defined areas in widely separated parts of the cord, are strong arguments in favour of the systematicness of the affection. (1), (2), and (5) together, make up what is usually regarded as Goll's column. (3), (4) and (6), constitute Burdach's column. The somewhat indefinite 'bandelettes' of Charcot probably correspond to (4).

Viewing the lesions in connection with the symptoms, this case seems to teach us (a) that sensory conduction does not take place either in the pyramidal strands, the cerebellar tracts, Goll's columns, or in the postero-external divisions of the posterior columns; (b) that with very extensive degeneration of the pyramidal tracts we may only have symptoms of paresis; and (c) that the posterior columns may be diseased in the lumbar region, and yet there be excessive tendon reflex.

In a second case, the symptoms were paralysis and contracture of the lower extremities, heightened tendon reflex, incontinence of urine, and a degree of analgesia in the legs. The autopsy gave degeneration of the lateral pyramidal tracts (particularly in the inferior dorsal and lumbar regions), the cerebellar tracts (degeneration most marked here), Goll's columns, and the postero-external divisions of the posterior columns. The anterior cornua were normal. The degenerated Goll's columns were traced to the upper part of the lumbar region.

Allusion is made to a third case, reported by Kahler and Pick,

in which the symptoms bore a strong resemblance to those observed in Friedreich's cases of hereditary ataxia, and where after death there was found systematic degeneration of the pyramidal strands, the cerebellar tracts, and Goll's columns. These three cases, Strümpell thinks, leave no doubt as to the existence of primary combined systematic diseases. The morbid process in these diseases is not a chronic inflammation originating in the connective tissue or blood-vessels, but is a true parenchymatous degeneration of the nerve fibres, the increase of connective tissue being secondary.

The paper closes with a case of tabes dorsalis, in which there was paralysis of the legs and ataxia and paresis of the upper extremities. The autopsy showed complete degeneration of the posterior columns with the exception of a small area adjoining the posterior commissure, and also systematic degeneration of the cerebellar tracts in their whole extent and of the lateral pyramidal strands in the lumbar and lower dorsal regions. A narrow belt of marginal degeneration surrounded the cord. The affection of the lateral columns in this case was too systematic to be accounted for on the usual theory of the direct extension of the disease from the posterior columns. The only feasible explanation is that tabes dorsalis belongs to the group of primary combined systematic diseases.

W. J. Dodds, D.Sc.

Syphilis and Tabes Dorsalis.—Westphal (*Archiv f. Psych.* xi. p. 230) concludes that an ætiological relation between syphilis and tabes is unsupported either by clinical or pathologico-anatomical facts. In 75 cases, whose histories were comparatively well ascertained, he found chancres in 14, chancres and secondary symptoms in 11. Nineteen out of 20 cases occurring in women had no history of chancre, and the twentieth case was a doubtful one. In one case only were secondary symptoms present whilst the patient was under observation. Of 16 cases that came to autopsy one only showed evidences of syphilis, and in another the appearances were doubtful. Further, Westphal has never seen a case of grey degeneration of the posterior columns cured by anti-syphilitic remedies. Remak (see *Centralblatt f. med. Wissensch.* 1880, No. 43) obtained a history of syphilis in 25 per cent. of his cases of tabes, nevertheless he does not admit a direct causal connection between the two. Bernhardt admits that syphilis may

produce tabes, but it is not nearly so potent a cause as is often supposed.

Westphal reports a case in which, besides other lesions of a syphilitic nature, there was disease of the posterior columns in the upper cervical region. The medulla of the nerves had disappeared, but the axis-cylinders were intact, and in some places seemed larger than normal. The vessels were dilated and their walls thickened. There was no increase of connective tissue or proliferation of nuclei, no corpora amylacea, no cells with fatty granules. The changes were evidently due to a peculiar parenchymatous affection. A gumma was found in the posterior part of the corpus callosum. Westphal compares this case with one recorded by Schultze, in which there was a tumour of the anterior part of the corpus callosum and degeneration of Burdach's columns down to the lower dorsal region. In Burdach's columns the nerves had lost their axis-cylinders, but for the most part retained their medulla. The neuroglia was normal.

Pick on Agenesis of the Spinal Cord. (*Prager med. Wochenschr.* 1880, Nos. 15 and 16, and *Centralblatt für med. Wissensch.* 1880. No. 46.)—The results of a *post-mortem* on a child aged 14 months are reported. There was hæmorrhagic pachymeningitis, asymmetry of the cerebrum and cerebellum (the former was smaller on the right side, the latter on the left), diminution of the crus, pons, and pyramid on the right side, and discoloration of the lateral pyramidal tracts as far down as the lower dorsal regions. The discoloured tract was smaller on the right side than on the left, but this was compensated for by a small tract of discoloration in the mesial part of the left anterior column, extending to the level of the fifth cervical nerve. The discoloration was due, not to hypertrophy of the interstitial connective tissue, but to the absence in most of the fibres of the medullary sheath. The strands had been arrested at a stage in their development prior to the appearance of the medullary substance. The author remarks that if the disease (encephalitis) which causes hemiatrophia cerebralis occurs in childhood or even youth when the cord is still developing, it is quite possible that an arrest of development may ensue in the corresponding pyramidal tracts.

Under the name of atelectasis medullæ spinalis, Schiff (*Pflügers Arch.* xxi. 328, and *Centralblatt f. med. Wissensch.*, No. 39), draws attention to a similar affection in the spinal cord of dogs. He describes several varieties; atelectasis totalis, segmentalis (medulla

of nerves wanting in particular strands), marginalis (medulla wanting in fibres that form border of cord), guttata and circinata (patches and rings in which fibres have no medulla). The axis-cylinders were quite intact, and the functions of the nerves apparently unimpaired. Schiff thinks that some of the cases of sclerosis and grey degeneration of the columns in man are really instances of a congenital affection of this kind; and in point of fact, he has found in general cases of the hereditary ataxia of Friedreich, not grey degeneration of the posterior columns, but simply an atelectasis. The retention of the sensibility in such cases is accounted for by the intactness of the axis-cylinders; the ataxia is a purely cerebral symptom.

Moeli on Amyotrophic Lateral Sclerosis.—The author reports a case of this disease (*Archiv f. Psych.* Bd. x. p. 718), which presents several interesting features. (1.) The patient died of pneumonia 3½ years after the appearance of the first symptoms, during which time no bulbar symptoms showed themselves. Cases have been known in which 6 and even 14 years intervened between the spinal and the bulbar symptoms. (2.) In the lower cervical and upper dorsal regions there was sclerosis of the mesial parts of Burdach's columns, such as is found in commencing tabes. There was no history of severe pains. (3.) There was no degeneration of the anterior columns: from which it follows that this was a case of total decussation of the pyramids. Total decussation appears unusually common in these cases. (4.) There was diminution of the irritability of the muscles, but the reaction of degeneration was never obtained. (5.) There were no muscular contractures. From this we may conclude that the cornua became diseased before the pyramidal tracts.

Shaw on Subacute Myelitis of the Anterior Cornua. (*Journ. Nerv. and Ment. Disease*, Chicago, April 1880.)—A woman, aged 50, as a result of a chill, became feverish, had pains in her chest, back, and feet, and lost the use of all her limbs. A month before death her condition was as follows: Complete paralysis of the upper extremities and atrophy of the muscles of the arms and forearms, able to move her feet a little, tactile sensibility of lower extremities slightly impaired; loss of faradic reaction in the muscles; occasional involuntary passage of urine and fæces. Bulbar symptoms appeared three days before death (about three months from commencement of illness), but patient was then semi-conscious. *Post-mortem:* there was a slight meningitis in the dorsal and

lumbar regions. Throughout the cord there was degeneration of the cells of the anterior cornua, especially of the external lateral group; many of the cells were swollen and cloudy, and had apparently lost their nuclei and processes. The grey matter in the neighbourhood of the cells showed, here and there, the granular disintegration of Lockhart Clarke. In the cervical enlargement, and in it only, there was sclerosis of the columns of Goll and of the lateral columns; in the upper half of the dorsal cord there was slight degeneration of the posterior root-zones (*rubans externes* of Charcot). The author thinks the ascending degeneration of Goll was secondary to the degeneration of the root-zones.

W. J. Dodds, D.Sc.

Erlenmeyer on Nerve-Stretching in Locomotor Ataxia. (*Centralblatt für Nervenheilkunde*, No. 21, 1880.)—Langenbuch and Esmarch have described two cases of tabes treated by nerve-stretching. The idea was to relieve the patients of their acute pains; and not only did the result justify the keenest hopes entertained on that score, but, to the astonishment of all concerned, the ataxy itself was greatly reduced by the operation. Erlenmeyer discusses these cases and their various bearings, and contributes another instance of nerve-stretching in an advanced case of tabes. The patient had passed through the period of shooting-pains, and had entered upon the third, or paralytical, stage of the disease. The ataxia was excessive, the muscular sense absolutely lost, the bladder paralysed. The most energetic treatment, including anti-syphilitic courses, had failed to effect any improvement. On June 22, 1880, the right sciatic nerve was exposed by means of an incision over the sciatic notches (Erlenmeyer considers it advisable to operate as near the centres as possible). Lister's method was followed, and the wound healed by first intention. On July 3, the left sciatic was stretched. At this time the right leg had distinctly gained in strength, but the ataxy was not notably diminished. The healing of the wound on the left side gave some trouble, on account of some erysipelatous complications. On the 16th of August, the patient could stand alone against a wall, which was previously impossible, and may be considered as a proof of a gain of strength. The ataxy remains much the same; but the remnants of the shooting-pains, which atmospheric vicissitudes always aroused, have disappeared.

The comparative failure in his case is attributed by Dr. Erlenmeyer to the insufficient force exerted in stretching the nerves.

But this method of treatment in locomotor ataxy, especially at an early stage, ought to be put to the test in a systematic manner. If possible the amount of traction necessary to produce the beneficial effects should be determined objectively by means of weights, or some other means of measurement.

Müller on the Early Stage of Locomotor Ataxy (reprint from the *Mittheilungen des Vereines der Aerzte in Steiermark*, 1879).—An early recognition of ataxic tabes is of the highest importance, as it is only at the earliest stage of the disease that we can hope to do much good by our remedial measures. The ocular symptoms are frequently the earliest; and among them the author draws attention to a sudden and transitory paralysis of accommodation of one eye. This may be the only trouble present, but is often accompanied with mydriasis paralytica. These symptoms may precede the actual invasion of the disease by some years. Then dissociated paralyses of the third, of the fourth, and sixth nerves may intervene, as well as spinal myosis, which is characterised by absence of reaction to light, but not to the accommodative impulse. Or, again, mere sluggishness or absence of reaction to light may be present. Charcot has well described the progressive optic atrophy; the papilla presents the appearance of a flat depression according to Müller, and the vessels gradually become smaller. The field of vision diminishes concentrically in the two eyes unequally, the region of dimness has the shape of sectors having (not the fovea, but) the papilla as centres; and this diminution is much more readily appreciated on indirect vision. Achromatopsia may occur, first to green, then to red. In disseminated sclerosis the sense of colour is preserved. The author recommends Doucet's perimeter as the most convenient to test these defects in practice.

The lancinating pains present various peculiarities which need not be dwelt upon here; the various other defects or alterations of sensibility are also well known. Berger has shown that there may be deficient algesia to powerful stimuli, whilst weaker ones are responded to. Retardation of transmission of sensations is not very common. The sense of pressure is often deficient at an early period.

The well-known "tendon reflex" may co-exist with ataxia (in 2 cases among 82: Berger). Certain facts lead the author to suspect a period of heightened reflex preceding its abolition.

[I have now under observation a somewhat doubtful case, in which there are shooting-pains in the left leg with very diminished tendon reflex, and markedly increased reflex in the right. The right hand is the seat of progressive muscular atrophy.]

Erlenmeyer has prominently brought forward the early gastric symptoms of tabes; they either take the form of nervous dyspepsia or actual catarrh. These symptoms have the characteristical suddenness of many tabetic symptoms.

The genito-urinary organs present numerous forms of disturbance. We may note here urethral and rectal neuralgias, causing spasmodic contraction of the bladder, and tenesmus.

The well-known crises gastriques may constitute for a long time the chief feature of the disease, and may require care in their diagnosis from other stomach ailments. The pneumogastric may be affected differently, and give rise to tremendous fits of coughing. The "crises néphritiques" need only be mentioned here.

Arthropathies and osseous fragility may appear early. The pulse is usually frequent. The sweat, increased at first, becomes deficient later on, and is not readily induced by pilocarpine in the diseased regions. The ataxic symptoms are usually pathognomic of the second period. The author has noticed two cases of early peroneal paralysis.

The treatment of tabes at this initial stage consists, first, in a strict hygienic mode of life; second, in the administration of ergot and nitrate of silver, a mild course of hydrotherapy (the water to range between 20°–25° centigrade) and electricity. The latter is to be applied centrally by means of large electrodes (2×6 inches), using a current strength of 8–11 milliwebers; and may be also usefully applied symptomatically for relieving the ocular troubles, the gastric pains, &c. Against the latter and the shooting-pains Chapman's bags may also be used with success.

The author next discusses the anti-syphilitic plan of treatment, and condemns it—first, because it is useless; second, because he does not consider the syphilitic nature of the disease as demonstrated. We have considered this point in our review of Vulpian's work (see page 523), where the main arguments of our author are briefly reproduced. Several instructive cases bring Dr. Müller's valuable paper to a close.

Erlenmeyer on the Paradoxical Muscular Contraction. (*Centralblatt Nervenheilk.* 1880, No. 17.)—Westphal has lately (*Archiv f. Psych.* x. 1) described a phenomenon under the name of "paradoxical muscular contraction," which occurs in certain neuroses of central origin, and consists in a contraction, or rather persistent contracture, of the tibialis anticus (and other muscles) when the foot is suddenly flexed. He gave the name "para-

doxical" under the assumption that the exciting cause of the contraction was the sudden relaxation of the muscle, and therefore was the opposite of that which occurs in the "tendon reflex," where the muscle reacts to a sudden tension exerted upon it. Erlenmeyer's observations fully confirm Westphal's description of the facts; but he dissents from the explanation given by the latter. He thinks that it is not the sudden relaxation of the muscle which is the cause of its contraction, but that the phenomenon is reflex, and that it is the sudden tension of the antagonistic calf muscles which excites the tibialis anticus to contract. In proof of this view Erlenmeyer says that if, whilst the contracture of the tibialis anticus is lasting, the calf muscles are pushed down with the hand so as to relax the tension exerted upon the tendo Achillis, the contractured muscle immediately returns to its normal condition. This takes place suddenly, not gradually. The experiment may be varied in this way—that the tendo Achillis is relaxed before the ankle is passively flexed: it is then observed that the "paradoxical contracture" does not occur. It is clear, therefore, that we have not to do here with the contraction produced directly by the sudden relaxation acting as a stimulant of the muscle, but as a spinal reflex, in which the sudden extension of a muscle acts as a stimulant of its antagonist. The phenomenon is one of great interest, and likely to throw light upon the question of tendon reflex generally. The author reminds the reader of the fact, recorded by Brissaud and Richet, that in patients suffering from hysterical contractures a vigorous extension of one muscle may bring about contracture of its antagonist, due apparently to a persistent irritation of the reflex medullary centre. [Westphal has since denied the value of E.'s test.]

Schulz on Unilateral Lesion of the Cord. (*Centralblatt für Nervenheilkunde*, No. 15, 1880.)—The author relates a case in which the spinal cord had been partially divided by a stab between the transverse processes of the fifth and sixth dorsal vertebræ on the right side. Immediately after the injury the patient suffered from anæsthesia of the left leg, hyperæsthesia and paresis of the right, with loss of power over the rectal sphincter, and retention of urine. He recovered partially, and about six months afterwards presented the following symptoms: No difference in the temperature and colour of the two legs. The muscular sense is diminished in the right along with the motility. There is no ataxy nor contracture, but the movements of the right leg lack power and precision.

There is marked hyperæsthesia on the right side from the seventh vertebra and rib downwards; above this a zone of anæsthesia between the sixth and seventh ribs. The sense of touch, and especially of tickling and of pain, is increased on the right, as well as the electrical excitability of the skin. On the left the sense of touch is preserved, but that of pain has disappeared. Tendon reflex much increased on the right. Electrical reactions equal on both sides. The functions of the rectum and bladder have resumed their normal course, and there is no disturbance in the sexual power.

In Gowers' case (*Lancet*, 1877, ii.) there was loss of tactile, as well as of algesic sense. In the present instance it is probable that the former was preserved, owing to the posterior tracts not being involved by the injury. Brown-Séquard's assumption that the paths for the muscular sense do not decussate in the cord is in accordance with the facts here observed. The author thinks that the phenomena now studied of transfer may throw some light upon the hitherto obscure symptom of hyperæsthesia observed in lateral lesions of the cord.

Dujardin-Beaumetz on Æsthesiogenic Properties of Certain Woods applied to the Skin. (*Bulletin de Thérapeutique*, Aug. 1880.)—It will be remembered that the main argument for reducing the supposed efficacity of metallic applications in hemi-anæsthesia to a mere effect of "expectant attention," was that in certain cases wooden discs were found to restore sensation as well as metal plates. At the time (*cf.* 'BRAIN,' IV.) I objected to this inference, that if the effect were due to statical effects of electricity the substitution of wood did not eliminate such an influence. It has since been found that a number of other agents besides discs and the like were equally active in hemi-anæsthesia, among which vibrating tuning-forks and the like. This fact led Prof. Schiff to assume that molecular vibrations induced in the nerve might explain the extraordinary effects observed (see *Archives de Physique*, Geneva, Jan. 1880; and the criticism by Vigouroux, *Progrès Médical*, 1880, No. 36). Whatever be the value of this hypothesis, the experiments of Dujardin-Beaumetz have shown not only that wood is an "æsthesiogenic" agent, but that this property varies greatly among the different kinds of wood. They were conducted with due care; and the numerous sources of error in such observations duly guarded against. The conclusions he arrives at are that woods vary greatly in their æsthesiogenic virtues; for instance, yellow cinchona is very active, more so indeed than metals, whilst

poplar, sycamore, palissander are inactive. Between the two rank rosewood, thuya, mahogany, walnut, &c.; but the list is as yet only provisionally given.

Berger on the Nosology of Tabes Dorsalis (*Centralblatt für Nervenheilkunde*, 1880, No. 5.)—Professor B. records the results of his observations on the behaviour of *cutaneous* sensibility in some cases of locomotor ataxia. This he found to reappear after its complete loss during the progress of the disease, and whilst the other symptoms were following the usual evolution. The restoration was remarkably complete (hyperalgesia even supervening), but was confined to the sensibility of the skin, and did not extend to the deeper parts. In one case a post-mortem examination revealed a complete posterior sclerosis; and the question naturally arises as to the path followed by the cutaneous impressions in the cord.

In one case of undoubted locomotor ataxia of 10 years' standing, (in which the characteristic lightning pains, the diplopia, bladder disturbance, sensory troubles, atactic gait, were present, along with the complete loss of tendon reflex), the latter symptom was investigated most carefully, so that there cannot be any doubt about the accuracy of the statement. After a few months, during which the patient took nitrate of silver and followed a hydrotherapic course, this general condition was found markedly improved. The symptoms taken singly had much abated; and the most remarkable feature about the case was a return of the patellar reflex, especially on the left side, where even a gentle tap would excite a most lively contraction of the triceps. The author has seen such a return of the tendon reflex in cases of diphtheritic ataxy, but does not think it has yet been noted in progressive ataxy, where it proves a possibility of at least a partial restoration of the cord.

Ballet on Facial Monoplegia (*Progrès Médical*, Sept. 1880).—A Salpétrière patient, female, age 71, sank suddenly to the ground, but did not lose consciousness. She was found to have lost all motor power over the left side of the face, without any corresponding paralysis of the arm and leg. During the day conjugate deviation to the right of the head and eyes set in; there was slight paresis of the left arm. Coma and death supervened.—Autopsy; right hemisphere: on inspection of the surface a cortical hemorrhage is seen, the size of a walnut, forming a clot of 5 grammes. It occupies the lower end of the

ascending frontal convolution, reaching below to the sylvian fissure. Methodical slicing shows that the inferior frontal fasciculus is destroyed, and the corresponding parietal fasciculus touched; but the central grey nuclei are intact.

A. DE WATTEVILLE.

Nitrite of Amyl in Epilepsy.—Dr. Maragliano (*Note di Clinica Medica*, Genoa, 1880) determined to try whether nitrite of amyl would so modify the cerebral circulation as to have a permanent effect in diminishing the fits of epilepsy. In inhaling the medicine, he used a bag of caoutchouc containing some cotton wool, which was applied to the nostrils while the patient was allowed to inspire air through the mouth. The dose given was much higher than that used by other physicians. Beginning at 10 drops he gradually mounted to 40, and he continued the inhalation as long as fifteen minutes without any instances in which harm was observed. Where the epileptic attacks were numerous, the inhalations were repeated once every hour. Dr. Maragliano quotes the experiments of Schüler, who found that in rabbits in which an opening had been made in the skull, from five to three inhalations caused a dilatation of the venous and arterial vessels of the pia mater. The smaller arteries began to pulsate, and some of them to assume serpentine curves. The brain expanded and bulged out of the trepanned hole. He notices the denial of Vulpian that nitrite of amyl has any congestive action on the brain, but finds from his own observation that the temperature of the head is increased by the action of this drug. In like manner he rejects the negative observations of Loemisch, Stammeshaus, and Pick for those of Aldridge, Tebaldi, and Gradenigo, who hold that the vessels of the retina are dilated after the use of inhalations of amyl. Ladendorf found that the temperature of the mouth was raised by half a degree C. during an inhalation. That this drug impedes the oxidation of the blood is universally admitted. He found that out of thirty cases after inhalation of amyl sugar appeared in the urine in twenty-four, and was totally absent in six. All traces of the sugar passed away entirely after three to six days. This appearance of sugar is supposed to be owing to the dilatation of the hepatic vessels. He admits that there are cases in which amyl is of no use in epilepsy, it promises most in those patients in whom the face becomes pale. Dr. Maragliano considers that the power this drug has in dilating the vessels of the brain (*la sua azione iperemizzante sul cervello*) is the cause of its therapeutic value in epilepsy.

Dr. Maragliano has made some very careful observations to ascertain the direct effect of the inspired nitrite of amyl upon the blood. He found that neither the size of the globules nor their number was affected by the drug.

The author sums up as follows:—

1. That the inhalation of nitrite of amyl methodically and persistently, and not with the view of cutting short impending attacks, causes a notable diminution of the violence and number of the epileptic seizures. This is proved by the three observations described, as well as several others in which the results were identical. The method employed by us is different from what is habitually followed, which was to cause the amyl to be inhaled after the beginning of the attack or after the aura came on in order to avert the fits. Hammond has tried the drug in much the same manner as ourselves without being able to report any benefit; but we using larger doses have obtained favourable results from the use of amyl. Some other observers have also reaped benefit, among them is Crichton-Browne, who however did not use the drug in the same fashion. He succeeded in eight cases out of ten in cutting short the status epilepticus in which fits succeeded one another without intermission, the patients remaining unconscious during the intervals.

2. That the dose of the nitrite of amyl may be raised without inconvenience to forty drops—the inhalations being prolonged for forty minutes and repeated from four to six times a day.

3. That the cerebral temperature is gradually raised from ·3 to ·8 of a degree C. during inhalation of the nitrites, on the cessation of which it again descends to the usual grade.

4. That the inhalation of nitrite of amyl in most instances causes the appearance of sugar in the urine amounting to from 1 to $2\frac{1}{2}$ grammes in the twenty-four hours.

W. W. Ireland.

END OF VOL. III.